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NEWS 3 MAY 08 CA/CAplus Indian patent publication number format defined
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NEWS 5 MAY 21 BIOSIS reloaded and enhanced with archival data
NEWS 6 MAY 21 TOXCENTER enhanced with BIOSIS reload
NEWS 7 MAY 21 CA/CAplus enhanced with additional kind codes for German patents
NEWS 8 MAY 22 CA/CAplus enhanced with IPC reclassification in Japanese patents
NEWS 9 JUN 27 CA/CAplus enhanced with pre-1967 CAS Registry Numbers
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NEWS 12 JUL 02 LEMBASE coverage updated
NEWS 13 JUL 02 LMEDLINE coverage updated
NEWS 14 JUL 02 SCISEARCH enhanced with complete author names
NEWS 15 JUL 02 CHEMCATS accession numbers revised
NEWS 16 JUL 02 CA/CAplus enhanced with utility model patents from China
NEWS 17 JUL 16 CAplus enhanced with French and German abstracts
NEWS 18 JUL 18 CA/CAplus patent coverage enhanced
NEWS 19 JUL 26 USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 20 JUL 30 USGENE now available on STN
NEWS 21 AUG 06 CAS REGISTRY enhanced with new experimental property tags
NEWS 22 AUG 06 BEILSTEIN updated with new compounds
NEWS 23 AUG 06 FSTA enhanced with new thesaurus edition

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

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=> s trap? (3a) vector
L1 696 TRAP? (3A) VECTOR

=> s l1 and ires and lox
L2 7 L1 AND IRES AND LOX

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 6 DUP REM L2 (1 DUPLICATE REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:530217 CAPLUS <<LOGINID::20070807>>
DN 144:1239
TI Characterization of an exchangeable gene trap using pU-17 carrying a stop codon-.beta.geo cassette
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi; Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, 862-0976, Japan
SO Development, Growth & Differentiation (2005), 47(3), 163-172
CODEN: DGDFA5; ISSN: 0012-1592
PB Blackwell Publishing Asia Pty Ltd.
DT Journal
LA English
AB We have developed anew exchangeable gene ***trap*** ***vector*** , pU-17, carrying the intron-lox71-splicing acceptor (SA)-.beta.-geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of .beta.galactosidase/neomycin-resistance fusion gene (.beta.geo) that can function in promoter trapping. We found that the ***trap*** ***vector*** was highly selective for integrations in the introns adjacent to the exon contg. the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the .beta.geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the .beta.geo gene. Thus, using this pU-17 ***trap*** ***vector*** , we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the .beta.geo gene with any gene of interest to be expressed under the control of the trapped promoter through Cre-mediated recombination.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout ***vector*** for gene ***trapping*** and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene trapping and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a splice acceptor sequence; (b) an internal ribosomal entry site (***IRES***) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 splice acceptor and polyadenylation signal, and human gene GTX element ***IRES*** . The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L3 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115

PRAI US 2001-336497P P 20011022
US 2002-196721 B2 20020715
US 2002-277612 A2 20021022

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene ***trap*** retroviral ***vector*** pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the splice acceptor in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x- ***IRES*** -DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the splice acceptor that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream ***IRES*** enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L3 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				

PRAI US 2001-330978P P 20011102
WO 2002-US35405 W 20021104

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary ***vector*** pDKO2 designed to ***trap*** transcriptionally active genes using the function of the splice acceptor in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x- ***IRES*** -DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.::retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. ***IRES*** enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:391889 CAPLUS <<LOGINID::20070807>>
DN 136:396975
TI Construction of recombinant vectors comprising a transcriptionally silent element for conditional gene inactivation in mammalian cells
IN Xin, Hong-Bo; Kotlikoff, Michael
PA Cornell Research Foundation, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040685	A2	20020523	WO 2001-US43916	20011116
WO 2002040685	A3	20030109		
WO 2002040685	A9	20030724		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002019841	A5	20020527	AU 2002-19841	20011116
US 2004077089	A1	20040422	US 2003-416995	20031106

PRAI US 2000-249200P P 20001116
WO 2001-US43916 W 20011116

AB The present invention provides recombinant vectors and methods of using the vectors in a high-throughput genetic system to rapidly generate conditional and/or conventional knockout mutants, such as mouse, useful to identify and define mammalian gene function in vivo. The methods of the invention combine gene trapping, gene targeting, and site-specific recombination techniques. The vectors comprise a transcriptionally silent genetic element that is inserted within a gene in a target cell in a manner that retains the functionality of the gene, and which element can be manipulated to inactivate the gene when desired. The vectors of the invention may be introduced ot cells via any means including non-biol. means, e.g., electroporation, or biol. means, e.g., via infection with a viral vector such as a retroviral vector.

L3 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene trap using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002

AB The gene trap technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of gene trapping is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene trap strategy. Here, we have constructed a new gene ***trap***

vector , pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (***IRES***)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the ***trap*** ***vector*** . By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the ***trap*** ***vector*** is lost through integration into the genome. Thus, this method, termed exchangeable gene trapping, has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

=> s l1 and splice acceptor
L4 59 L1 AND SPLICE ACCEPTOR

=> s l4 and lox
L5 4 L4 AND LOX

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 4 DUP REM L5 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout ***vector*** for gene ***trapping*** and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene trapping and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a ***splice*** ***acceptor*** sequence; (b) an internal ribosomal entry site (IRES) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 ***splice*** ***acceptor*** and polyadenylation signal, and human gene GTX element IRES. The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L6 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene ***trap*** retroviral ***vector*** pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the ***splice*** ***acceptor*** in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x-IRES-DR-bGHpA (S/A: branch site and ***splice***. ***acceptor*** from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the ***splice*** ***acceptor*** that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream IRES enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L6 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary ***vector*** pDKO2 designed to ***trap***

transcriptionally active genes using the function of the ***splice*** ***acceptor*** in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x-IRES-DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and ***splice*** ***acceptor*** (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the ***splice*** ***acceptor*** provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. IRES enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L6 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:238349 CAPLUS <<LOGINID::20070807>>
DN 138:216479
TI method for selecting a clone of a cell library containing mutation introduced by gene trapping
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO Eur. Pat. Appl., 17 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1295951	A1	20030326	EP 2001-308108	20010924

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRAI EP 2001-308108 20010924
AB This invention provides a method for selecting a clone of a cell library contg. a mutation in a gene that is expressed in a test cell. The method comprising: providing cDNA obtained by reverse transcription of mRNA of the test cell; providing a collection of cultured cells of said library organized into individual clones, wherein each clone is of a cell having a mutation in an exon in its genome, the mutation being in a different exon in cells of different clones; providing an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations in; exposing the cDNA to the array under conditions permitting hybridization of polynucleotides in the array to nucleic acids; detecting hybridization of cDNA to a polynucleotide on the array; and selecting a clone in the collection from which a hybridizing polynucleotide detected at is an exon fragment. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon ***trap*** ***vector*** for mutating ES cells.
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 19:09:44 ON 07 AUG 2007)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:09:57 ON 07 AUG 2007
L1 696 S TRAP? (3A) VECTOR
L2 7 S L1 AND IRES AND LOX
L3 6 DUP REM L2 (1 DUPLICATE REMOVED)
L4 59 S L1 AND SPLICE ACCEPTOR
L5 4 S L4 AND LOX
L6 4 DUP REM L5 (0 DUPLICATES REMOVED)

=> s l1 and lox
L7 14 L1 AND LOX

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 11 DUP REM L7 (3 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 11 ANSWERS - CONTINUE? Y/(N):y

L8 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:405865 CAPLUS <<LOGINID::20070807>>
DN 144:481709
TI Exchangeable gene trapping
AU Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kumamoto, Japan
SO Genetically Engineered Mice Handbook (2006), 131-142. Editor(s): Sundberg, John P.; Ichiki, Tsutomu. Publisher: CRC Press LLC, Boca Raton, Fla.
CODEN: 69IBMO; ISBN: 978-0-8493-2220-4
DT Conference; General Review
LA English
AB A review discusses the principle, advantages, and disadvantages of gene trapping, with emphasis on the Cre-mutant ***Lox*** site-specific integration system and the pU-17 exchangeable ***trap*** ***vector*** .
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 11 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2005:248868 BIOSIS <<LOGINID::20070807>>
DN PREV200510036473
TI Characterization of an exchangeable gene trap using pU-17 carrying a stop codon-beta geo cassette.
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi [Reprint Author]; Kimi, Araki
CS Kumamoto Univ, Inst Mol Embryol and Genet, Kuhonji 4-24-1, Kumamoto 8620976, Japan
yamamura@gpo.kumamoto-u.ac.jp; arakimi@gpo.kumamoto-u.ac.jp
SO Development Growth & Differentiation, (APR 2005) Vol. 47, No. 3, pp. 163-172.
CODEN: DGDFA5. ISSN: 0012-1592.
DT Article
LA English
ED Entered STN: 8 Jul 2005
Last Updated on STN: 8 Jul 2005
AB We have developed a new exchangeable gene ***trap*** ***vector*** pU-17, carrying the intron-lox71-splicing acceptor (SA)-beta geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of beta galactosidase/neomycin-resistance fusion gene (beta geo) that can function in promoter trapping. We found that the ***trap*** ***vector*** was highly selective for integrations in the introns adjacent to the exon containing the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the beta geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the beta geo gene. Thus, using this pU-17 ***trap*** ***vector*** , we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the beta geo gene with any gene of interest to be expressed under the control of the trapped promoter through Cre-mediated recombination.

L8 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout ***vector*** for gene ***trapping*** and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530

PRAI US 2003-448395 A 20030530
AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene trapping and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a splice acceptor sequence; (b) an internal ribosomal entry site (IRES) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 splice acceptor and polyadenylation signal, and human gene GTX element IRES. The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L8 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English

FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene ***trap*** retroviral ***vector*** pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the splice acceptor in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x-IRES-DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the splice acceptor that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream IRES enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L8 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary ***vector*** pDKO2 designed to ***trap*** transcriptionally active genes using the function of the splice acceptor in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x-IRES-DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. IRES enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L8 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:238349 CAPLUS <<LOGINID::20070807>>
DN 138:216479
TI method for selecting a clone of a cell library containing mutation introduced by gene trapping
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO Eur. Pat. Appl., 17 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1295951	A1	20030326	EP 2001-308108	20010924
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
PRAI EP 2001-308108		20010924		

AB This invention provides a method for selecting a clone of a cell library contg. a mutation in a gene that is expressed in a test cell. The method comprising: providing cDNA obtained by reverse transcription of mRNA of the test cell; providing a collection of cultured cells of said library organized into individual clones, wherein each clone is of a cell having a mutation in an exon in its genome, the mutation being in a different exon in cells of different clones; providing an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations in; exposing the cDNA to the array under conditions permitting hybridization of polynucleotides in the array to nucleic acids; detecting hybridization of cDNA to a polynucleotide on the array; and selecting a clone in the collection from which a hybridizing polynucleotide detected at is an exon fragment. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon ***trap*** ***vector*** for mutating ES cells.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:391889 CAPLUS <<LOGINID::20070807>>
DN 136:396975
TI Construction of recombinant vectors comprising a transcriptionally silent element for conditional gene inactivation in mammalian cells
IN Xin, Hong-Bo; Kotlikoff, Michael
PA Cornell Research Foundation, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040685	A2	20020523	WO 2001-US43916	20011116
WO 2002040685	A3	20030109		
WO 2002040685	A9	20030724		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,				

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002019841 A5 20020527 AU 2002-19841 20011116
US 2004077089 A1 20040422 US 2003-416995 20031106
PRAI US 2000-249200P P 20001116
WO 2001-US43916 W 20011116

AB The present invention provides recombinant vectors and methods of using the vectors in a high-throughput genetic system to rapidly generate conditional and/or conventional knockout mutants, such as mouse, useful to identify and define mammalian gene function in vivo. The methods of the invention combine gene trapping, gene targeting, and site-specific recombination techniques. The vectors comprise a transcriptionally silent genetic element that is inserted within a gene in a target cell in a manner that retains the functionality of the gene, and which element can be manipulated to inactivate the gene when desired. The vectors of the invention may be introduced ot cells via any means including non-biol. means, e.g., electroporation, or biol. means, e.g., via infection with a viral vector such as a retroviral vector.

L8 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:961831 CAPLUS <<LOGINID::20070807>>
DN 138:249000
TI Large scale gene trap mutagenesis
AU Araki, Masatake; Araki, Kimi
CS Institute of Genetic Experiment, Kumamoto University, Japan
SO Igaku no Ayumi (2002), 203(3), 201-205
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese

AB A review. The gene trap method for mutagenesis of embryonic stem cells by using promoter-less trap vectors was discussed. The outlines of the strategies of gene targeting were described by covering the topics on vector design and establishing transgenic mice lines. The genetic substitution by the trap technique and prodn. gene knockin mice and control of the transgene expression by the Cre/ ***lox*** system were also described. The impact of application of the trap method to the large scale prodn: of transgenic animal models for human diseases was discussed.

L8 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:924281 CAPLUS <<LOGINID::20070807>>
DN 136:32674
TI Embryonic stem cell libraries indexed to nucleic acid microarrays
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2001053524	A1	20011220	US 2001-883745	20010618
US 6867035	B2	20050315		
CA 2309371	A1	20011216	CA 2000-2309371	20000616
CA 2350976	A1	20011216	CA 2001-2350976	20010618

PRAI CA 2000-2309371 A 20000616

AB The invention relates to libraries of embryonic stem cells in which the genome of members of the library are modified by gene trapping. This invention provides a method for selecting a clone of an ES cell contg. a mutation in a gene that is expressed in a test cell. The method comprising, obtaining cDNA of the test cell, providing a collection of cultured ES cells organized into individual clones, wherein each clone of an ES cell having a mutation in an exon in its genome. The invention further provides an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations; exposing the cDNA to the array under conditions permitting hybridization of polynucleotide. The invention relates to detection of hybridization of cDNA to a polynucleotide on the array and selecting a clone in the collection. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon ***trap*** ***vector*** for mutating ES cells.

RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 10 OF 11 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 2
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene trap using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002

AB The gene trap technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of gene trapping is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene trap strategy. Here, we have constructed a new gene ***trap*** ***vector*** , pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (IRES)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the ***trap*** ***vector*** . By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the ***trap*** ***vector*** is lost through integration into the genome. Thus, this method, termed exchangeable gene trapping, has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

L8 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:390639 CAPLUS <<LOGINID::20070807>>
DN 125:50249
TI Trapping of mammalian promoters by cre- ***lox*** site-specific recombination
AU Fukushige, Shinichi; Ikeda, Joh-E.
CS Ikeda GenoSPHERE Project, ERATO, Japan
SO DNA Research (1996), 3(2), 73-80
CODEN: DARSE8; ISSN: 1340-2838
PB Kazusa DNA Research Institute
DT Journal
LA English

AB One of the challenges in human genome research is to identify the promoter sequences which play a key role in the regulation of gene expression. We report here a new promoter trapping system for use with mammalian cells comprised of the following three steps: (1) Cloning of DNA fragments into a promoter- ***trapping*** ***vector*** , (2) integration of the ***trapping*** ***vector*** into a designated target in the mammalian genome using the Cre-site-specific recombinase, and (3) screening of integrants for trapped promoter sequences by activation of the luciferase gene. To assess the efficiency of this system, ***lox*** trapping vectors contg. sense tk promoter, antisense tk promoter, or a non-promoter sequence of the neo gene were employed. The resulting levels of luciferase activity of the site specific integrants were measured directly. Luciferase activity of the integrants can be assayed under conventional culture conditions by simply replacing the culture medium with potassium phosphate buffer contg. luciferin. Only those G418-resistant colonies carrying the tk promoter in the normal orientation exhibited a 21-35-fold increase in luciferase activity over that of the other integrants. These results indicate that this system is an effective means of trapping promoter sequences from random mammalian genomic DNA fragments.

=> FIL STNGUIDE

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		83.32	83.53

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		-14.04	-14.04

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FILE CONTAINS CURRENT INFORMATION.
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=> s trap? and vector
L9 4930 TRAP? AND VECTOR

=> s l9 and lox
L10 20 L9 AND LOX

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11 15 DUP REM L10 (5 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 15 ANSWERS - CONTINUE? Y/(N):y

L11 ANSWER 1 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:405865 CAPLUS <<LOGINID::20070807>>
DN 144:481709
TI Exchangeable gene ***trapping***
AU Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kumamoto, Japan
SO Genetically Engineered Mice Handbook (2006), 131-142. Editor(s): Sundberg, John P.; Ichiki, Tsutomu. Publisher: CRC Press LLC, Boca Raton, Fla.
CODEN: 69IBMO; ISBN: 978-0-8493-2220-4
DT Conference; General Review
LA English
AB A review discusses the principle, advantages, and disadvantages of gene ***trapping*** , with emphasis on the Cre-mutant ***Lox*** site-specific integration system and the pU-17 exchangeable ***trap*** ***vector*** .
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2005:248868 BIOSIS <<LOGINID::20070807>>
DN PREV200510036473
TI Characterization of an exchangeable gene ***trap*** using pU-17 carrying a stop codon-beta geo cassette.
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi [Reprint Author]; Kimi, Araki
CS Kumamoto Univ, Inst Mol Embryol and Genet, Kuhonji 4-24-1, Kumamoto 8620976, Japan
yamamura@gpo.kumamoto-u.ac.jp; arakimi@gpo.kumamoto-u.ac.jp
SO Development Growth & Differentiation, (APR 2005) Vol. 47, No. 3, pp. 163-172.
CODEN: DGDFA5. ISSN: 0012-1592.
DT Article
LA English
ED Entered STN: 8 Jul 2005
Last Updated on STN: 8 Jul 2005
AB We have developed a new exchangeable gene ***trap*** ***vector*** , pU-17, carrying the intron-lox71-splicing acceptor (SA)-beta geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of beta galactosidase/neomycin-resistance fusion gene (beta geo) that can function in promoter ***trapping*** . We found that the ***trap*** ***vector*** was highly selective for integrations in the introns adjacent to the exon containing the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the beta geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the beta geo gene. Thus, using this pU-17 ***trap*** ***vector*** , we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the beta geo gene with any gene of interest to be expressed under the control of the ***trapped*** promoter through Cre-mediated recombination.

L11 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:109968 CAPLUS <<LOGINID::20070807>>
DN 142:349687
TI Gene ***trap*** and gene inversion methods for conditional gene inactivation in the mouse
AU Xin, Hong-Bo; Deng, Ke-Yu; Shui, Bo; Qu, Shimian; Sun, Qi; Lee, Jane; Greene, Kai Su; Wilson, Jason; Yu, Ying; Feldman, Morris; Kotlikoff, Michael I.
CS Department of Biomedical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY, 14853, USA
SO Nucleic Acids Research (2005), 33(2), e14/1-e14/10
CODEN: NARHAD; ISSN: 0305-1048
PB Oxford University Press
DT Journal
LA English
AB Conditional inactivation of individual genes in mice using site-specific recombinases is an extremely powerful method for detg. the complex roles of mammalian genes in developmental and tissue-specific contexts, a major goal of post-genomic research. However, the process of generating mice with recombinase recognition sequences placed at specific locations within a gene, while maintaining a functional allele, is time consuming, expensive and tech. challenging. We describe a system that combines gene ***trap*** and site-specific DNA inversion to generate mouse embryonic stem (ES) cell clones for the rapid prodn. of conditional knockout mice, and the use of this system in an initial gene ***trap*** screen. Gene ***trapping*** should allow the selection of thousands of ES cell clones with defined insertions that can be used to generate conditional knockout mice, thereby providing extensive parallelism that eliminates the time-consuming steps of targeting ***vector*** construction and homologous recombination for each gene.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout ***vector*** for gene ***trapping*** and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene ***trapping*** and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a splice acceptor sequence; (b) an internal ribosomal entry site (IRES) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 splice acceptor and polyadenylation signal, and human gene GTX element IRES. The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L11 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene ***trap*** vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof)

and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene ***trap*** retroviral ***vector*** pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to ***trap*** transcriptional active genes using the function of the splice acceptor in the ***vector*** and the ***Lox*** signals-flanked gene ***trap*** portion contains S/A-x-IRES-DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the ***vector*** is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the ***trapped*** endogenous gene onto the splice acceptor that is provided by the ***vector*** . Once this splicing event occurs, a fusion transcript will be made that results in the introduction of ***vector*** encoded stop codons that cause the premature termination of the protein product of the ***trapped*** gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream IRES enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene ***trap*** efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L11 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral ***vector*** insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

PRAI US 2001-330978P P 20011102
WO 2002-US35405 W 20021104

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary ***vector*** pDKO2 designed to ***trap*** transcriptionally active genes using the function of the splice acceptor in the ***vector*** is described. PDKO2 (3'LTR- ***lox*** -S/A-x-IRES-DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains ***vector*** backbone from self-inactivating retroviral ***vector*** pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the ***vector*** is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the ***vector*** . Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the ***trapped*** gene. IRES enables the expression of the drug selection marker when an active promoter is ***trapped*** , which allows selection of gene ***trap*** event. The retrovirus produced from .RetroPack PT67 cells is used to infect Jurkat and gene ***trapping*** is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L11 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:238349 CAPLUS <<LOGINID::20070807>>
DN 138:216479
TI method for selecting a clone of a cell library containing mutation introduced by gene ***trapping***
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO Eur. Pat. Appl., 17 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1295951	A1	20030326	EP 2001-308108	20010924

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRAI EP 2001-308108 20010924

AB This invention provides a method for selecting a clone of a cell library contg. a mutation in a gene that is expressed in a test cell. The method comprising: providing cDNA obtained by reverse transcription of mRNA of the test cell; providing a collection of cultured cells of said library organized into individual clones, wherein each clone is of a cell having a mutation in an exon in its genome, the mutation being in a different exon in cells of different clones; providing an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations in; exposing the cDNA to the array under conditions permitting hybridization of polynucleotides in the array to nucleic acids; detecting hybridization of cDNA to a polynucleotide on the array; and selecting a clone in the collection from which a hybridizing polynucleotide detected at is an exon fragment. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon ***trap*** ***vector*** for mutating ES cells.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
AN 2003:240214 BIOSIS <<LOGINID::20070807>>
DN PREV200300240214
TI Generation and genetic modification of dendritic cells derived from mouse embryonic stem cells.
AU Senju, Satoru; Hirata, Shinya; Matsuyoshi, Hidetake; Masuda, Masako; Uemura, Yasushi; Araki, Kimi; Yamamura, Ken-Ichi; Nishimura, Yasuharu [Reprint Author]
CS Division of Immunogenetics, Department of Neuroscience and Immunology, Graduate School of Medical Sciences, Kumamoto University, 2-2-1 Honjo, Kumamoto, 860-0811, Japan
mxnishim@gpo.kumamoto-u.ac.jp
SO Blood, (May1 2003) Vol. 101, No. 9, pp. 3501-3508. print.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 21 May 2003
Last Updated on STN: 21 May 2003

AB We developed a method to generate dendritic cells (DCs) from mouse embryonic stem (ES) cells. We cultured ES cells for 10 days on feeder cell layers of OP9, in the presence of granulocyte-macrophage colony-stimulating factor in the latter 5 days. The resultant ES cell-derived cells were transferred to bacteriologic Petri dishes without feeder cells and further cultured. In about 7 days, irregularly shaped floating cells with protrusions appeared and these expressed major histocompatibility complex class II, CD11c, CD80, and CD86, with the capacity to stimulate primary mixed lymphocyte reaction (MLR) and to process and present protein antigen to T cells. We designated them ES-DCs (ES cell-derived dendritic cells), and the functions of ES-DCs were comparable with those of DCs generated from bone marrow cells. Upon transfer to new dishes and stimulation with interleukin-4 plus tumor necrosis factor alpha, combined with anti-CD40 monoclonal antibody or lipopolysaccharide, ES-DCs completely became mature DCs, characterized by a typical morphology and higher capacity to stimulate MLR. Using an expression ***vector*** containing the internal ribosomal entry site-puromycin N-acetyltransferase gene or a Cre- ***lox*** -mediated exchangeable gene- ***trap*** system, we could efficiently generate ES cell transfectants expressing the products of introduced genes after their differentiation to DCs. ES-DCs expressing invariant chain fused to a pigeon cytochrome C epitope presented the epitope efficiently in the context of Ek. We primed ovalbumin (OVA)-specific cytotoxic T lymphocytes

in vivo by injecting mice with ES-DCs expressing OVA, thus demonstrating immunization with ES-DCs genetically engineered to express antigenic protein. The methods may be applicable to immunomodulation therapy and gene- ***trap*** investigations of DCs.

L11 ANSWER 9 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:391889 CAPLUS <<LOGINID::20070807>>
DN 136:396975
TI Construction of recombinant vectors comprising a transcriptionally silent element for conditional gene inactivation in mammalian cells
IN Xin, Hong-Bo; Kotlikoff, Michael
PA Cornell Research Foundation, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040685	A2	20020523	WO 2001-US43916	20011116
WO 2002040685	A3	20030109		
WO 2002040685	A9	20030724		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002019841	A5	20020527	AU 2002-19841	20011116
US 2004077089	A1	20040422	US 2003-416995	20031106
PRAI US 2000-249200P	P	20001116		
WO 2001-US43916	W	20011116		

AB The present invention provides recombinant vectors and methods of using the vectors in a high-throughput genetic system to rapidly generate conditional and/or conventional knockout mutants, such as mouse, useful to identify and define mammalian gene function in vivo. The methods of the invention combine gene ***trapping*** , gene targeting, and site-specific recombination techniques. The vectors comprise a transcriptionally silent genetic element that is inserted within a gene in a target cell in a manner that retains the functionality of the gene, and which element can be manipulated to inactivate the gene when desired. The vectors of the invention may be introduced ot cells via any means including non-biol. means, e.g., electroporation, or biol. means, e.g., via infection with a viral ***vector*** such as a retroviral ***vector*** .

L11 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:961831 CAPLUS <<LOGINID::20070807>>
DN 138:249000
TI Large scale gene ***trap*** mutagenesis
AU Araki, Masatake; Araki, Kimi
CS Institute of Genetic Experiment, Kumamoto University, Japan
SO Igaku no Ayumi (2002), 203(3), 201-205
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A review. The gene ***trap*** method for mutagenesis of embryonic stem cells by using promoter-less ***trap*** vectors was discussed. The outlines of the strategies of gene targeting were described by covering the topics on ***vector*** design and establishing transgenic mice lines. The genetic substitution by the ***trap*** technique and prodn. gene knockin mice and control of the transgene expression by the Cre/ ***lox*** system were also described. The impact of application of the ***trap*** method to the large scale prodn. of transgenic animal models for human diseases was discussed.

L11 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:924281 CAPLUS <<LOGINID::20070807>>
DN 136:32674
TI Embryonic stem cell libraries indexed to nucleic acid microarrays
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2001053524	A1	20011220	US 2001-883745	20010618
US 6867035	B2	20050315		
CA 2309371	A1	20011216	CA 2000-2309371	20000616
CA 2350976	A1	20011216	CA 2001-2350976	20010618
PRAI CA 2000-2309371	A	20000616		

AB The invention relates to libraries of embryonic stem cells in which the genome of members of the library are modified by gene ***trapping*** . This invention provides a method for selecting a clone of an ES cell contg. a mutation in a gene that is expressed in a test cell. The method comprising, obtaining cDNA of the test cell, providing a collection of cultured ES cells organized into individual clones, wherein each clone of an ES cell having a mutation in an exon in its genome. The invention further provides an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations; exposing the cDNA to the array under conditions permitting hybridization of polynucleotide. The invention relates to detection of hybridization of cDNA to a polynucleotide on the array and selecting a clone in the collection. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon ***trap*** ***vector*** for mutating ES cells.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:648874 CAPLUS <<LOGINID::20070807>>
DN 136:242836
TI Balanced-Size and Long-Size Cloning of Full-Length, Cap- ***Trapped*** cDNAs into Vectors of the Novel .lambda.-FLC Family Allows Enhanced Gene Discovery Rate and Functional Analysis
AU Carninci, Piero; Shibata, Yuko; Hayatsu, Norihito; Itoh, Masayoshi; Shiraki, Toshiyuki; Hirozane, Tomoko; Watahiki, Akira; Shibata, Kazuhiro; Konno, Hideaki; Muramatsu, Masami; Hayashizaki, Yoshihide
CS Genome Exploration Research Group, RIKEN Genomic Sciences Center (GSC), Suehiro-cho, Tsurumi-ku, Yokohama, Kanagawa, 230-0045, Japan
SO Genomics (2001), 77(1-2), 79-90
CODEN: GNMCEP; ISSN: 0888-7543
PB Academic Press
DT Journal
LA English
AB We have developed a new class of cloning vectors: .lambda.-full-length cDNA (.lambda.-FLC) cloning vectors. These vectors can be bulk-excised for prepg. full-length cDNA libraries in which a high proportion of the plasmids carry large inserts that can be transferred into other (for example, functional) vectors. Unlike other cloning vectors, .lambda.-FLC vectors accommodate a broad range of sizes of eukaryotic cDNA inserts because they contain "size balancers. " Further, the main protocol we use for direct bulk excision of plasmids is mediated by a Cre- ***lox*** system and is apparently free of size bias. The av. size of the inserts from excised plasmid cDNA libraries was 2.9 kb for std. and 6.9 kb for size-selected cDNA. The av. insert size of the full-length cDNA libraries was correlated to the rate of new gene discovery, suggesting that effectively cloning rarely expressed mRNAs requires vectors that can accommodate large inserts from a variety of sources. Part of the vectors are also suitable for bulk transfer of inserts into various functional vectors. (c) 2001 Academic Press.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 13 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 3
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene ***trap*** using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB The gene ***trap*** technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of gene ***trapping*** is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene ***trap*** strategy. Here, we have constructed a new gene ***trap*** ***vector*** , pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (IRES)-beta-geo-pA-loxP-pA-pUC. By using this ***vector*** , we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 ***trap*** clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the ***trap*** ***vector*** . By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid ***vector*** sequence can be introduced even when the pUC sequence of the ***trap*** ***vector*** is lost through integration into the genome. Thus, this method, termed exchangeable gene ***trapping*** ,

has many advantages as the ***trapped*** clones can be utilized to express genes with any type of mutation.

L11 ANSWER 14 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1998:486316 BIOSIS <<LOGINID::20070807>>
DN PREV199800486316
TI Linoleic acid peroxidation by Solanum tuberosum lipoxygenase was activated in the presence of human 5-lipoxygenase-activating protein.
AU Battu, Serge [Reprint author]; Moalic, Sandra; Rigaud, Michel; Beneytout, Jean-Louis
CS Lab. Biochimie, Fac. Med. Pharm. 2 rue Docteur Marcland, 87025 Limoges Cedex, France
SO Biochimica et Biophysica Acta, (June 15, 1998) Vol. 1392, No. 2-3, pp. 340-350. print.
CODEN: BBACAQ. ISSN: 0006-3002.
DT Article
LA English
ED Entered STN: 5 Nov 1998
Last Updated on STN: 5 Nov 1998
AB The present investigation describes the ability of human 5-lipoxygenase-activating protein (FLAP) to activate a plant 5-lipoxygenase. The presence of an active recombinant human FLAP in the 100,000 X g membrane fraction of infected Sf9 cells led to a specific increase in 9-hydroperoxyoctadecadienoic acid (9-HPOD) synthesis (+68%) or in 5-hydroperoxyeicosatetraenoic acid (5-HPETE) synthesis (+68%), after action of Solanum tuberosum tuber 5-lipoxygenase (S. t. ***LOX***) on linoleic acid (natural plant lipoxygenase substrate) or on arachidonic acid. On the contrary, the presence of non-transfected membranes obtained from non-infected Sf9 cells led to an inhibition of lipoxygenase activity. MK-886, a potent inhibitor of leukotriene biosynthesis, blocked the FLAP dependent S. t. ***LOX*** activation after preincubation with FLAP transfected membranes. In conclusion, this study demonstrates that a recombinant human FLAP can stimulate a lipoxygenase other than mammalian 5-lipoxygenase (S. t. ***LOX***) by using different polyunsaturated fatty acids as substrates.

L11 ANSWER 15 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:390639 CAPLUS <<LOGINID::20070807>>
DN 125:50249
TI ***Trapping*** of mammalian promoters by cre- ***lox*** site-specific recombination
AU Fukushige, Shinichi; Ikeda, Joh-E.
CS Ikeda GenoSPHERE Project, ERATO, Japan
SO DNA Research (1996), 3(2), 73-80
CODEN: DARSE8; ISSN: 1340-2838
PB Kazusa DNA Research Institute
DT Journal
LA English
AB One of the challenges in human genome research is to identify the promoter sequences which play a key role in the regulation of gene expression. We report here a new promoter ***trapping*** system for use with mammalian cells comprised of the following three steps: (1) Cloning of DNA fragments into a promoter- ***trapping*** ***vector*** , (2) integration of the ***trapping*** ***vector*** into a designated target in the mammalian genome using the Cre-site-specific recombinase, and (3) screening of integrants for ***trapped*** promoter sequences by activation of the luciferase gene. To assess the efficiency of this system, ***lox*** ***trapping*** vectors contg. sense tk promoter, antisense tk promoter, or a non-promoter sequence of the neo gene were employed. The resulting levels of luciferase activity of the site specific integrants were measured directly. Luciferase activity of the integrants can be assayed under conventional culture conditions by simply replacing the culture medium with potassium phosphate buffer contg. luciferin. Only those G418-resistant colonies carrying the tk promoter in the normal orientation exhibited a 21-35-fold increase in luciferase activity over that of the other integrants. These results indicate that this system is an effective means of ***trapping*** promoter sequences from random mammalian genomic DNA fragments.

=> s insertion? (3a)(mutation or mutagen?)
L12 21182 INSERTION? (3A)(MUTATION OR MUTAGEN?)

=> s l13 and lox
L13 NOT FOUND
The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l12 and lox
L13 30 L12 AND LOX

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14 23 DUP REM L13 (7 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 23 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1226337 CAPLUS <<LOGINID::20070807>>
DN 146:1620
TI PiggyBac transposon as a tool for genetic manipulation and analysis in vertebrates
IN Xu, Tian; Han, Min; Zhuang, Yuan; Wu, Xiaohui; Ding, Sheng; Li, Gang
PA Fudan University, Peop. Rep. China
SO PCT Int. Appl., 114pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006122442	A1	20061123	WO 2005-CN674	20050514

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI WO 2005-CN674 20050514
AB The present invention is based on the discovery that piggyBac, a DNA transposon from the cabbage looper moth Trichoplusia ni, can transpose efficiently in vertebrate, including mammalian, cells, both in vivo and ex vivo. PiggyBac transposition occurs almost exclusively at TTAA sites following a precise cut-and-paste manner. When introduced into fertilized eggs the piggyBac transposon can integrate into the mouse genome without obvious chromosome regional preferences, and preferably inserted into transcriptional units. Also, piggyBac elements can carry multiple marker genes and allow the expression of these genes at various insertion sites. Thus, the piggyBac transposon system, and other members of the "piggyBac-like" transposon family, are valuable new tools for efficient genetic manipulation and anal. in mice and other vertebrates, including transgenesis and ***insertional*** ***mutagenesis*** .
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2006:241118 BIOSIS <<LOGINID::20070807>>
DN PREV200600236940
TI Human endogenous retrovirus HERV-H family in human tissues and cancer cells: expression, identification, and phylogeny.
AU Yi, Joo-Mi; Kim, Hwan-Mook; Kim, Heui-Soo [Reprint Author]
CS Pusan Natl Univ, Coll Nat Sci, Div Biol Sci, Pusan 609735, South Korea
khs307@pusan.ac.kr
SO Cancer Letters, (JAN 18 2006) Vol. 231, No. 2, pp. 228-239.
CODEN: CALEDQ. ISSN: 0304-3835.
DT Article
LA English
ED Entered STN: 19 Apr 2006
Last Updated on STN: 19 Apr 2006
AB HERV-H family is the most abundant HERV families in the human genome with more than 1000 copies including full-length, truncated form, and solitary LTRs. We investigated envelope (env) gene fragments of HERV-H family in various human tissues and cancer cells. The em, fragments were detected in mRNA of several human tissues (placenta, skeletal muscle, spleen, and thymus) and cancer cells (RT4, BT-474, HCT-116 TE-1, UO-31, Jurkat, HepG2, A549, MCF7, OVCAR-3, MIA-PaCa-2, PC3, ***LOX*** -IMVI, AZ521, 2F7, U-937, and C-33A) by RT-PCR approach. The RT-PCR products were cloned and sequenced. New 12 clones from human tissues and 48 clones from cancer cells of env gene sequences belonging to the HERV-H family showed 84.3-98.1% sequence similarity to that of HERV-H (AF108843). Deduced amino acid sequences of 60 clones from human tissues and cancer cell lines showed multiple frameshifts and termination codons caused by deletion/ ***insertion*** or point ***mutation*** with the exception of eight clones as following: HHE9-1. HHE9-5 (skeletal muscle), HHE10-5 (spleen), CHE10-9 (MCF7), CHE12-4, CHE12-5 (MIA-PaCa-2), and CHE18-1, CHE18-3 (C-33A) to that of HERV-H (AF108843). A phylogenetic tree of the HERV-H family was constructed to understand their relationship, indicating that they were divided into three groups, one major (group I) and two minor (group II and III), through sequence divergence. The HERV-H families in group I has been proliferated on human genome during hominoid evolution. These active HERV-H elements are worthy of further investigations as potential pathogenic effects to various human diseases including cancers. (c) 2005 Elsevier Ireland Ltd. All rights reserved.

L14 ANSWER 3 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
AN 2006:335071 BIOSIS <<LOGINID::20070807>>
DN PREV200600329128
TI High efficiency site-specific genetic engineering of the mosquito genome.
AU Nimmo, D. D.; Alphey, L.; Meredith, J. M.; Eggleston, P. [Reprint Author]
CS Univ Keele, Sch Life Sci, Ctr Appl Entomol and Parasitol, Huxley Bldg,

Keele ST5 5BG, Staffs, UK
bia35@keele.ac.uk
SO Insect Molecular Biology, (APR 2006) Vol. 15, No. 2, pp. 129-136. ISSN: 0962-1075.
DT Article
LA English
ED Entered STN: 28 Jun 2006
Last Updated on STN: 28 Jun 2006
AB Current techniques for the genetic engineering of insect genomes utilize transposable genetic elements, which are inefficient, have limited carrying capacity and give rise to position effects and ***insertional*** ***mutagenesis*** . As an alternative, we investigated two site-specific integration mechanisms in the yellow fever mosquito, Aedes aegypti. One was a modified CRE/ .***lox*** system from phage P1 and the other a viral integrase system from Streptomyces phage phi C31. The modified CRE/ ***lox*** system consistently failed to produce stable germline transformants but the phi C31 system was highly successful, increasing integration efficiency by up to 7.9-fold. The ability to efficiently target transgenes to specific chromosomal locations and the potential to integrate very large transgenes has broad applicability to research on many medically and economically important species.

L14 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:293460 CAPLUS <<LOGINID::20070807>>
DN 140:282414
TI Methods for ends-out or replacement gene targeting in Drosophila
IN Golic, Kent G.; Gong, Wei J.
PA USA
SO U.S. Pat. Appl. Publ., 27 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004068761	A1	20040408	US 2003-434668	20030509
PRAI US 2002-416561P	P	20021007		

AB Ends-in and ends-out refer to the two arrangements of donor DNA that can be used for gene targeting. Both have been used for targeted mutagenesis, but require donors of differing design. Ends-out targeting is more frequently used in mice and yeast because it gives a straightforward route to replace or delete a target locus. Although ends-in targeting has been successful in Drosophila, an attempt at ends-out targeting failed. To test whether ends-out targeting could be used in Drosophila two strategies for ends-out gene replacement at the endogenous yellow (y) locus in Drosophila were applied. First, a mutant allele was rescued by replacement with an 8-kb y+ DNA fragment at a rate of .apprxeq.1/800 gametes. Second, a wild-type gene was disrupted by the insertion of a marker gene in exon 1 at a rate of .apprxeq.1/380 gametes. The I-SceI endonuclease component alone is not sufficient for targeting and the FLP recombinase is also needed to generate the extrachromosomal donor. When both components are used, ends-out targeting can be approx. as efficient as ends-in targeting, and is likely to be generally useful for Drosophila gene targeting.

L14 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one ***mutagen*** is an ***insertional*** ***mutagen*** , to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by ***insertional*** ***mutagenesis*** is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene trap retroviral vector pDKO2 (also called ***insertional*** ***mutagen***) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the splice acceptor in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x-IRES-DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the splice acceptor that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream IRES enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L14 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:837330 CAPLUS <<LOGINID::20070807>>
DN 141:290040
TI Methods for inducible site-directed mutagenesis through conditional gene rescue
IN Gotthardt, Michael; Radke, Michael
PA Max-Delbrueck-Centrum fuer Molekulare Medizin, Germany
SO Eur. Pat. Appl., 19 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1466974	A1	20041013	EP 2003-8470	20030411
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
WO 2004090131	A1	20041021	WO 2004-EP2216	20040304
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2006242720	A1	20061026	US 2006-551658	20060622
PRAI EP 2003-8470	A	20030411		
WO 2004-EP2216	W	20040304		

AB The present invention relates to methods for inducible site-directed mutagenesis through conditional gene rescue. The mutated allele of said gene interferes with survival and/or causes an adverse phenotype, such as temporal and/or local phenotypes, such as cell cycle-specific, cell-type specific, tissue-specific, protein-expression specific, tissue-development specific, organ-specific, organ-development-specific and/or embryonic lethal phenotypes. According to further aspects thereof, the present invention relates to a conditionally inducible site-directed mutant cell culture, tissue, organ, or non-human embryo, comprising a cell and a resp. non-human organism, in particular a genetically deficient or Knock-out-mammal, -rodent, -nematode, -fish, -plant or -insect. Finally the invention provides a method for inducible site-directed mutagenesis through conditional gene rescue, either in vitro or in vivo.

L14 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:109915 CAPLUS <<LOGINID::20070807>>
DN 140:265446
TI The region 3' to Xist mediates X chromosome counting and H3 Lys-4 dimethylation within the Xist gene
AU Morey, Celine; Navarro, Pablo; Debrand, Emmanuel; Avner, Philip; Rougeulle, Claire; Clerc, Philippe
CS Genetique Moleculaire Murine, Institut Pasteur, Paris, Fr.
SO EMBO Journal (2004), 23(3), 594-604
CODEN: EMJODG; ISSN: 0261-4189
PB Nature Publishing Group
DT Journal
LA English
AB A counting process senses the X chromosome/autosome ratio and ensures that
X chromosome inactivation (XCI) initiates in the female (XX) but not in the male (XY) mouse embryo. Counting is regulated by the X-inactivation center, which contains the Xist gene. Deleting 65 kb 3' to Xist in XO embryonic stem (ES) cells affects counting and results in inappropriate

XCI upon differentiation. We show here that normal counting can be rescued in these deleted ES cells using cre/loxP re-insertion, and refine the location of elements controlling counting within a 20 kb bipartite domain. Furthermore, we show that the 65 kb deletion also leads to inappropriate XCI in XY differentiated ES cells, which excludes the involvement of sex-specific mechanisms in the initiation of XCI. At the chromatin level, we have found that the Xist gene corresponds to a peak of H3 Lys-4 dimethylation, which is dramatically and specifically affected by the deletion 3' to Xist. Our results raise the possibility that H3 Lys-4 dimethylation within Xist may be functionally implicated in the counting process.
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:837254 CAPLUS <<LOGINID::20070807>>
DN 139:302989
TI VAC-BAC shuttle vector system comprising modified vaccinia virus and use for gene expression
IN Moss, Bernard; Domi, Arban
PA Government of the United States of America, as Represented by the Secretary Department of Health and Human Services, USA
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003087330	A2	20031023	WO 2003-US11183	20030410
WO 2003087330	A3	20040325		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003221885	A1	20031027	AU 2003-221885	20030410
EP 1495125	A2	20050112	EP 2003-718343	20030410
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2005124070	A1	20050609	US 2004-959392	20041005
PRAI US 2002-371840P	P	20020410		
US 2002-402824P	P	20020809		
WO 2003-US11183	W	20030410		

AB The invention relates to a VAC-BAC shuttle vector system for creation of recombinant poxviruses from DNA cloned in a bacterial artificial chromosome. The VAC-BAC vector system contains a vaccinia virus genome (VAC) that can replicate in bacteria and produce infectious virus in mammalian cells. The VAC-BAC vector system can be used to modify vaccinia virus DNA by deletion, ***insertion*** or point ***mutation*** or add new DNA to the VAC genome with methods developed for bacterial plasmids, rather than by recombination in mammalian cells. It can also be used to produce recombinant vaccinia viruses for gene expression and prodn. of modified vaccinia viruses that have improved safety or immunogenicity.

L14 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector ***insertional*** ***mutagenesis***
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as ***insertional*** ***mutagens*** . Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the ***insertional*** ***mutagen*** (s). The ***insertional*** ***mutagen*** thus allows identification of one or more genes that are mutated by ***insertion*** of an ***insertional*** ***mutagen*** . The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an ***insertional*** ***mutagen*** incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the ***insertional*** ***mutagens*** . The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary vector pDKO2 designed to trap transcriptionally active genes using the function of the splice acceptor in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x-IRES-DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, IRES: wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. IRES enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L14 ANSWER 10 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:140664 BIOSIS <<LOGINID::20070807>>
DN PREV200400133839
TI Identification of Cbfb-MYH11 cooperating genes in acute myeloid leukemia using a conditional Cbfb-MYH11 knock-in BXH-2 mouse model.
AU Kuo, Ya-Huei [Reprint Author]; Perrat, Paola [Reprint Author]; Martinez, Natalia [Reprint Author]; Castilla, Lucio H. [Reprint Author]
CS Program in Gene Function and Expression, Medical School, University of Massachusetts, Worcester, MA, USA
SO Blood, (November 16 2003) Vol. 102, No. 11, pp. 367a. print.
Meeting Info.: 45th Annual Meeting of the American Society of Hematology. San Diego, CA, USA. December 06-09, 2003. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004

AB The chromosome 16 inversion is one of the most frequent chromosome rearrangements found in human acute myeloid leukemia (AML). This inversion disrupts CBFB and MYH11, creating the fusion gene CBFB-MYH11. Previous studies using a Cbfb-MYH11 knock-in mouse model showed that Cbfb-MYH11 blocks embryonic definitive hematopoiesis and adult hematopoietic differentiation towards myeloid and lymphoid lineages. Moreover, Cbfb-MYH11 predisposes mice to AML but is not sufficient for leukemogenesis. To facilitate further studies of Cbfb-MYH11 mediated AML, we created a Cbfb-MYH11 conditional knock-in model (CbfbloxCbfb-MYH11), using the Cre-loxP recombination system. These mice are leukemia free, indicating that the targeted Cbfb allele does not express Cbfb-MYH11. Also, homozygous CbfbloxCbfb-MYH11 developed normally, suggesting that Cbfb expressed from the targeted Cbfb allele is functional. To efficiently induce Cbfb-MYH11 expression in the adult bone marrow, we crossed CbfbloxCbfb-MYH11/+ and Mx1-Cre transgenic mice. Over 80% of the bone marrow cells from CbfbloxCbfb-MYH11/+/Mx1-Cre mice had efficiently deleted sequences between the ***lox*** sites one week after pI-pC injection shown by Southern blot analysis. As previously shown with Cbfb-MYH11 knock-in chimeras, CbfbloxCbfb-MYH11/+/Mx1-Cre mice treated with pI-pC survived more than a year without leukemia. To identify Cbfb-MYH11 cooperating genes by retrovirus ***insertional*** ***mutagenesis*** (RIM), CbfbloxCbfb-MYH11/+/Mx1-Cre were introduced into BXH-2 background through a series of backcrosses. BXH-2 mice spontaneously produce high titers of B-ecotropic murine leukemia virus and succumb to AML or lymphoma between 6 and 10 months of age. Recent RIM

studies in BXH-2 mice have identified more than 2000 integration sites, offering powerful genetic system to carry out large scale screening for leukemia associated genes. In our study, Cbfb-MYH11 significantly accelerates the onset of AML in first-, second- and third-generation BXH-2 backcrosses (F1, F2 and F3). About 75% of CbfbloxCbfb-MYH11/+/Mx1-Cre mice in BXH-2 F1 developed AML 4 to 6 months after induction of Cbfb-MYH11 expression. In contrast, control BXH-2 F1 mice survived leukemia-free up to one year of age (experimental endpoint). CbfbloxCbfb-MYH11/+/Mx1-Cre mice in BXH-2 F2 and F3 have developed AML 3 to 5 months after the induction of Cbfb-MYH11 expression with 100% penetrance whereas the control BXH-2 F2 mice are leukemia-free up to 9 to 12 months of age. Finally, all Cbfb-MYH11 expressing mice analyzed developed AML with myelomonocytic morphology, while no lymphoma was detected. These results support the hypothesis that Cbfb-MYH11 provides a strong sensitizing background and accelerates exclusively the onset of AML in BXH-2 mice. Inverse PCR followed by sequencing and genome database analyses are currently underway to identify candidate genes that cooperate with Cbfb-MYH11 in the tumors we have obtained to date. Candidate cooperating genes isolated will be discussed.

L14 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:946895 CAPLUS <<LOGINID::20070807>>
DN 140:298511
TI Transpositional behavior of an Ac/Ds system for reverse genetics in rice
AU Greco, R.; Ouwerkerk, P. B. F.; De Kam, R. J.; Sallaud, C.; Favalli, C.; Colombo, L.; Guiderdoni, E.; Meijer, A. H.; Hoge, J. H. C.; Pereira, A.
CS Plant Research International, Wageningen, 6700 AA, Neth.
SO Theoretical and Applied Genetics (2003), 108(1), 10-24
CODEN: THAGA6; ISSN: 0040-5752
PB Springer-Verlag
DT Journal
LA English
AB A collection of transposon Ac/Ds enhancer trap lines is being developed in rice that will contribute to the development of a rice mutation machine for the functional anal. of rice genes. Mol. analyses revealed high transpositional activity in early generations, with 62% of the T0 primary transformants and more than 90% of their T1 progeny lines showing ongoing active transposition. About 10% of the lines displayed amplification of the Ds copy no. However, inactivation of Ds seemed to occur in about 70% of the T2 families and in the T3 generation. Southern blot analyses revealed a high frequency of germinal insertions inherited in the T1 progeny plants, and transmitted preferentially over the many other somatic inserts to later generations. The sequencing of Ds flanking sites in subsets of T1 plants indicated the independence of insertions in different T1 families originating from the same T0 line. Almost 80% of the insertion sites isolated showing homol. to the sequenced genome, resided in genes or within a range at which neighboring genes could be revealed by enhancer trapping. A strategy involving the propagation of a large no. of T0 and T1 independent lines is being pursued to ensure the recovery of a max. no. of independent insertions in later generations. The inactive T2 and T3 lines produced will then provide a collection of stable insertions to be used in reverse genetics expts. The preferential insertion of Ds in gene-rich regions and the use of lines contg. multiple Ds transposons will enable the prodn. of a large population of inserts in a smaller no. of plants. Addnl. features provided by the presence of ***lox*** sites for site-specific recombination, or the use of different transposase sources and selectable markers, are discussed.
RE.CNT 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:461222 CAPLUS <<LOGINID::20070807>>
DN 137:42547
TI Mismatch repair detection applicable for high-throughput genotyping and mutation detection
IN Cox, David R.; Faham, Malek; Baharloo, Siamak
PA The Board of Trustees of the Leland Stanford Junior University, USA; The Regents of the University of California
SO U.S., 18 pp., Cont.-in-part of U.S. Ser. No. 713,751, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6406847	B1	20020618	US 1999-271055	19990317
CA 2365123	A1	20000921	CA 2000-2365123	20000314
WO 2000055369	A1	20000921	WO 2000-US6731	20000314
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1175510	A1	20020130	EP 2000-916338	20000314
EP 1175510	B1	20070228		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY				
JP 2002538840	T	20021119	JP 2000-605785	20000314
AU 780056	B2	20050224	AU 2000-37458	20000314
AT 355388	T	20060315	AT 2000-916338	20000314
US 2002172966	A1	20021121	US 2002-72047	20020208
US 6709827	B2	20040323		
US 2003003472	A1	20030102	US 2002-81771	20020220
US 7153652	B2	20061226		
US 2005239080	A1	20051027	US 2003-728122	20031203
PRAI US 1995-4664P	P	19951002		
US 1996-713751	B2	19960913		
US 1999-271055	A	19990317		
WO 2000-US6731	W	20000314		
US 2002-72047	A2	20020208		

AB Mismatch Repair Detection (MRD), a novel method for DNA-variation detection, utilizes bacteria to detect mismatches by a change in expression of a marker gene. DNA fragments to be screened for variation are cloned into two MRD plasmids, and bacteria are transformed with heteroduplexes of these constructs. Resulting colonies express the marker gene in the absence of a mismatch, and lack expression in the presence of a mismatch. MRD is capable of detecting a single mismatch within 10 kb of DNA. In addn., MRD can analyze many fragments simultaneously, offering a powerful method for high-throughput genotyping and mutation detection. The method was demonstrated using two puC-derived plasmids, pMF200 and pMF100, which are identical except that there is a 5-bp insertion in the lacZ.alpha. gene of pMF100. The method of mutation detection comprises cloning one copy of the DNA in question in pMF200, the other copy into pMF100. The pMF200 plasmid is cloned in dam- E. coli (no methylation of the plasmid); the pMF100 plasmid is cloned in dam+ E. coli (methylation of plasmid). The plasmids are isolated, linearized, denatured, and reannealed, then digested with MboI and DpnI. E. coli are transformed with the resulting hemimethylated heteroduplexes. The transformants are cultured and .beta.-galactosidase activity detected as usual. If no mutation was present (i.e., no mismatch), no repair occurs and the colonies are blue. If a mutation was present, repair occurs and the lacZ.alpha. mutant is corepaired resulting in colonies with white color. In addn. of LacZ.alpha. gene, the gene for Cre recombinase (Cre) can be also used as the marker gene for bacteria carrying two antibiotic selection markers (tetR and strepS genes) flanked by two ***lox*** sites. The resulting colonies are tetracycline sensitive and streptomycin resistant in the absence of a mismatch and tetracycline resistant and streptomycin sensitive in the presence of a mismatch.
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 13 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN
AN 2002:387097 BIOSIS <<LOGINID::20070807>>
DN PREV200200387097
TI Allele ***insertion*** ***mutagenesis*** at the EP3 receptor locus.
AU Breyer, Richard M. [Reprint author]; Defoe, Stephanie K. [Reprint author]; Breyer, Matthew D. [Reprint author]; Lee, Daekee; Young-Seigler, Artenzia C.; Threadgill, David
CS Medicine, Vanderbilt University, 1161 21st Ave South, S3223 MCN, Nashville, TN, 37232-2372, USA
SO FASEB Journal, (March 20, 2002) Vol. 16, No. 4, pp. A576. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Jul 2002
Last Updated on STN: 17 Jul 2002
AB Prostaglandin E2 acts via 4 G-protein coupled E-prostanoid (EP) receptors. Three EP3 receptor splice-variants have been identified in mice, alpha, beta, and gamma. To evaluate the role of the EP3 receptor splice variants in vivo, a mouse "knock-in" strategy that limits the expressed EP3 repertoire to a single splice variant was employed. A 6 kb fragment of the EP3 receptor gene was cloned and sequenced. This fragment contains the second coding exon (TMVI and TMVII) as well as the contiguous gamma exon. A vector replacing the gamma exon with a beta exon was targeted into ES cells by homologous recombination. The exon 2/beta fusion was flanked with ***lox*** P sites to permit subsequent Cre mediated deletion of the target allele. Cre electroporation of correctly targeted ES cells resulted in the deletion of the EP3 exon and resulted in a single remnant loxP71 site. These ES cells were co-transfected a third time with a Cre expressing plasmid and a pBluescript vector into which a variant loxP site (loxP66) and a mutant EP3 receptor had been subcloned. The EP3 alpha allele was successfully inserted into the EP3 locus by Cre mediated recombination. This provides a platform for the facile manipulation of the EP3 locus.

L14 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:542924 CAPLUS <<LOGINID::20070807>>
DN 138:33810
TI ***Insertional*** ***mutagenesis*** : transposon- ***insertion*** libraries as ***mutagens*** in yeast
AU Kumar, Anuj; Vidan, Susana; Snyder, Michael
CS Dep. of Mol., Cellular, and Dev. Biol., Yale Univ., New Haven, CT, 06520, USA
SO Methods in Enzymology (2002), 350(Guide to Yeast Genetics and Molecular and Cell Biology, Part B), 219-229

CODEN: MENZAU; ISSN: 0076-6879
PB Academic Press
DT Journal
LA English
AB Comprehensive protocols for using ***insertional*** libraries as ***mutagens*** in yeast are provided. A plasmid-based library of transposon-mutagenized yeast DNA is used to generate and identify the target yeast mutants. By employing insertional libraries carrying a specially designed multipurpose transposon, insertions are modified in yeast to generate corresponding epitope-tagged alleles for various functional studies. (c) 2002 Academic Press.
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 15 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005445137 EMBASE <<LOGINID::20070807>>
TI Functional genomics in the mouse.
AU Perkins A.S.
CS A.S. Perkins, Department of Pathology and Molecular, Cellular, Developmental Biology, Yale University, P.O. Box 208023, New Haven, CT 06520-8023, United States. archibald.perkins@yale.edu
SO Functional and Integrative Genomics, (2002) Vol. 2, No. 3, pp. 81-91. .
Refs: 65
ISSN: 1438-793X CODEN: FIGUBY
CY Germany
DT Journal; General Review
FS 022 Human Genetics
LA English
SL English
ED Entered STN: 28 Nov 2005
Last Updated on STN: 28 Nov 2005
AB The mouse is the premier genetic model organism for the study of human disease and development. With the recent advances in sequencing of the human and mouse genomes, there is strong interest now in large-scale approaches to decipher the function of mouse genes using various mutagenesis technologies. This review discusses what tools are currently available for manipulating and mutagenizing the mouse genome, such as ethylnitrosourea and gene trap mutagenesis, engineered inversions and deletions using the cre- ***lox*** system, and proviral ***insertional*** ***mutagenesis*** in somatic cells, and how these are being used to uncover gene function. .COPYRGT. Springer-Verlag 2002.

L14 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:319910 CAPLUS <<LOGINID::20070807>>
DN 134:336659
TI Transgenic mice containing disruptions in trp gene encoding trinucleotide repeat proteins and uses
IN Klein, Robert; Matthews, William; Moore, Mark; Allen, Keith D.
PA Deltagen, Inc., USA
SO PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001030798	A1	20010503	WO 2000-US29382	20001026
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2388192	A1	20010503	CA 2000-2388192	20001026
AU 200113435	A	20010508	AU 2001-13435	20001026
EP 1224199	A1	20020724	EP 2000-975372	20001026
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
BR 2000015100	A	20030715	BR 2000-15100	20001026
JP 2003531573	T	20031028	JP 2001-533149	20001026
MX 2002PA04175	A	20030820	MX 2002-PA4175	20020426
ZA 2002003820	A	20030904	ZA 2002-3820	20020514
PRAI US 1999-161488P	P	19991026		
WO 2000-US29382	W	20001026		

AB The present invention relates to transgenic animals, compns. and methods relating to the characterization of gene function, and more specifically to genes encoding trinucleotide repeat proteins (TRP). The invention provides a knockout mouse having a non-functional allele for the gene that naturally encodes and expresses a functional TRP. One aspect of the invention is a knockout mouse having a phenotype including reduced wt., or bone disease, or cartilage disease, or kidney disease. The present invention provides a method of identifying agents capable of affecting a phenotype of a knockout mouse. The invention provides novel genes encoding TRPs, including T243 gene.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:666920 CAPLUS <<LOGINID::20070807>>
DN 133:248033
TI Mismatch repair detection utilizing bacteria to detect mismatches by a change in expression of a marker gene
IN Cox, David R.; Faham, Malek; Baharloo, Siamak
PA The Board of Trustees of the Leland Stanford Junior University, USA; The Regents of the University of California
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000055369	A1	20000921	WO 2000-US6731	20000314
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6406847	B1	20020618	US 1999-271055	19990317
CA 2365123	A1	20000921	CA 2000-2365123	20000314
EP 1175510	A1	20020130	EP 2000-916338	20000314
EP 1175510	B1	20070228		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY				
JP 2002538840	T	20021119	JP 2000-605785	20000314
AU 780056	B2	20050224	AU 2000-37458	20000314
PRAI US 1999-271055	A	19990317		
US 1995-4664P	P	19951002		
US 1996-713751	B2	19960913		
WO 2000-US6731	W	20000314		

AB Mismatch Repair Detection (MRD), a novel method for DNA-variation detection, utilizes bacteria to detect mismatches by a change in expression of a marker gene. DNA fragments to be screened for variation are cloned into two MRD plasmids, and bacteria are transformed with heteroduplexes of these constructs. Resulting colonies express the marker gene in the absence of a mismatch, and lack expression in the presence of a mismatch. MRD is capable of detecting a single mismatch within 10 kb of DNA. In addn., MRD can analyze many fragments simultaneously, offering a powerful method for high-throughput genotyping and mutation detection. Mismatch Repair Detection (MRD), a novel method for DNA-variation detection, utilizes bacteria to detect mismatches by a change in expression of a marker gene. DNA fragments to be screened for variation are loned into two MRD plasmids, and bacteria are transformed with heteroduplexes of these constructs. Resulting colonies express the marker gene in the absence and a mismatch, and lack expression in the presence of a mismatch. MRD is capable of detecting a single mismatch within 10 kb of DNA. In addn., MRD has the potential for analyzing many fragments simultaneously, offering a powerful method for high-throughput genotyping and mutation detection in a large genomic region. The method was demonstrated using two puC-derived plasmids, pMF200 and pMF100, which are identical except that there is a 5-bp insertion in the lacZ.alpha. gene of pMF100. The method of mutation detection comprises cloning one copy of the DNA in question in pMF200, the other copy into pMF100. The pMF200 plasmid is cloned in dam- Escherichia coli (no methylation of the plasmid); the pMF100 plasmid is cloned in dam+ E. coli (methylation of plasmid). The plasmids are isolated, linearized, denatured, and reannealed, then digested with MboI and DpnI. E. coli are transformed with the resulting hemimethylated heteroduplexes. The transformants are cultured and .beta.-galactosidase activity detected as usual. If no mutation was present (i.e., no mismatch), no repair occurs and the colonies are blue. If a mutation was present, repair occurs and the lacZ.alpha. mutant is corepaired resulting in colonies with white color.
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 18 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:291273 CAPLUS <<LOGINID::20070807>>
DN 132:318600
TI Targeted gene modification by gene conversion using parvovirus vectors
IN Russell, David W.; Hirata, Roli K.
PA University of Washington, USA
SO PCT Int. Appl., 90 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000024917	A1	20000504	WO 1999-US25462	19991027
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
CA 2348778	A1	20000504	CA 1999-2348778	19991027
GB 2358865	A	20010808	GB 2001-10672	19991027

DE 19983694 T0 20020529 DE 1999-19983694 19991027
JP 2002528088 T 20020903 JP 2000-578469 19991027
PRAI US 1998-106191P P 19981028
WO 1999-US25462 W 19991027
AB This invention provides methods for obtaining targeted gene modification in vertebrate cells by a gene conversion-like process using parvoviral vectors, including adeno-assocd. virus (AAV). The parvoviral vectors used in the methods of the invention are capable of targeting a specific genetic modification to a preselected target locus in a cellular genome by homologous pairing. Use of an adeno-assocd. virus 2 vector to correct a 14 base pair insertion in a neomycin phosphotransferase gene is demonstrated. Similarly, the induction of an ***insertion*** ***mutation*** in the human hypoxanthine phosphoribosyltransferase (HPRT) gene was demonstrated in HeLa cells. The rate of mutation to 6-thioguanine resistance was increased about 30-fold over control cells infected with vector without the HPRT gene sequence. Comparable results were seen using normal human fibroblasts.
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:414404 CAPLUS <<LOGINID::20070807>>
DN 131:197138
TI Molecular analysis of a null mutant for pea (Pisum sativum L.) seed lipoxygenase-2
AU Forster, Colette; North, Helen; Afzal, Naureen; Domoney, Claire; Hornostaj, Andrzej; Robinson, David S.; Casey, Rod
CS John Innes Centre, Norwich, NR4 7UH, UK
SO Plant Molecular Biology (1999), 39(6), 1209-1220
CODEN: PMBIDB; ISSN: 0167-4412
PB Kluwer Academic Publishers
DT Journal
LA English
AB A mutant line of Pisum fulvum was identified that lacked seed lipoxygenase-2 (***LOX*** -2). The mutant phenotype was introgressed into a std. Pisum sativum cv. Birte to provide near-isogenic lines with or without seed ***LOX*** -2. Genetic analyses showed the mutation to behave as a single, recessive Mendelian gene. Northern and dot blot analyses showed a large redn. in ***LOX*** -2 mRNA from developing seeds of the ***LOX*** -2-null mutant. A restriction fragment length polymorphism assocd. with the 5' end of the ***LOX*** -2 gene(s) co-segregated with the null phenotype, indicating that the redn. of ***LOX*** -2 mRNA was neither a consequence of deletion of the ***LOX*** genes nor a consequence of the action of a genetically distant regulatory gene. Anal. of the 5'-flanking sequences of ***LOX*** -2 genes from Birte and the near-isogenic ***LOX*** -2-null mutant revealed a no. of insertions, deletions and substitutions within the promoter from the ***LOX*** -2-null mutant that could be responsible for the null phenotype. Incubation of crude seed ***LOX*** prepns. from Birte and the ***LOX*** -2-null mutant showed that the latter generated relatively less 13-hydroperoxides and also produced relatively more hydroxy- and ketoacid compds. that have implications for the fresh-frozen pea industry.
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 20 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 3
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene trap using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB The gene trap technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of gene trapping is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene trap strategy. Here, we have constructed a new gene trap vector, pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (IRES)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random ***insertional*** ***mutagenesis*** as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the trap vector. By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the trap vector is lost through integration into the genome. Thus, this method, termed exchangeable gene trapping, has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

L14 ANSWER 21 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 4
AN 1998:302298 BIOSIS <<LOGINID::20070807>>
DN PREV199800302298
TI Effects of procollagen C-proteinase enhancer protein on the growth of cultured rat fibroblasts revealed by an excisable retroviral vector.
AU Masuda, Michiaki [Reprint author]; Igarashi, Hiroko; Kano, Munehide; Yoshikura, Hiroshi
CS Dep. Microbiol., Grad. Sch. Med., Univ. Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113, Japan
SO Cell Growth and Differentiation, (May, 1998) Vol. 9, No. 5, pp. 381-391. print.
ISSN: 1044-9523.
DT Article
LA English
ED Entered STN: 15 Jul 1998
Last Updated on STN: 13 Aug 1998
AB An excisable retroviral vector, TSN- ***lox*** , was developed by exploiting Cre-loxP homologous recombination. An integrated TSN- ***lox*** provirus could be excised, leaving a solo long terminal repeat at the integration site; inverse PCR, taking advantage of the solo long terminal repeat, was used to characterize cellular flanking sequences. A TSN- ***lox*** -transduced Rat2 cell clone, ***lox*** -7, was found to harbor the provirus in an intron of the procollagen C-proteinase enhancer protein (PCPE) gene, whose expression was lowered compared with that of the parental Rat2. When the vector provirus in ***lox*** -7 cells was excised, PCPE expression was elevated. The level of PCPE expression seemed to affect cell growth properties such as morphology, contact inhibition, and anchorage-independent growth. These results suggested that the excisable retroviral vector may be useful for studying the molecular basis for proviral ***insertion*** ***mutagenesis*** , and that PCPE may play a significant role in controlling cell growth and differentiation.

L14 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1997:39297 CAPLUS <<LOGINID::20070807>>
DN 126:140261
TI A multipurpose transposon system for analyzing protein production, localization, and function in Saccharomyces cerevisiae
AU Ross-Macdonald, Petra; Sheehan, Amy; Roeder, G. Shirleen; Snyder, Michael
CS Dep. Biol., Yale Univ., New Haven, CT, 06520-8103, USA
SO Proceedings of the National Academy of Sciences of the United States of America (1997), 94(1), 190-195
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
AB Anal. of the function of a particular gene product typically involves detg. the expression profile of the gene, the subcellular location of the protein, and the phenotype of a null strain lacking the protein. Conditional alleles of the gene are often created as an addnl. tool. The authors have developed a multifunctional, transposon-based system that simultaneously generates constructs for all the above analyses and is suitable for mutagenesis of any given Saccharomyces cerevisiae gene. Depending on the transposon used, the yeast gene is fused to a coding region for .beta.-galactosidase or green fluorescent protein. Gene expression can therefore be monitored by chem. or fluorescence assays. The transposons create insertion mutations in the target gene, allowing phenotypic anal. The transposon can be reduced by cre- ***lox*** site-specific recombination to a smaller element that leaves an epitope tag inserted in the encoded protein. In addn. to its utility for a variety of immunodetection purposes, the epitope tag element also has the potential to create conditional alleles of the target gene. The authors demonstrate these features of the transposons by mutagenesis of the SPA2, ARP100, SER1, and BDF1 genes.
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 23 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 5
AN 1995:267037 BIOSIS <<LOGINID::20070807>>
DN PREV199598281337
TI A system for ***insertional*** ***mutagenesis*** and chromosomal rearrangement using the Ds transposon and Cre-lox.
AU Osborne, Brian I.; Wirtz, Uwe; Baker, Barbara [Reprint author]
CS Plant Gene Expression Center, Univ. California, Berkeley USDA-ARS, 800 Buchanan St., Albany, CA 94710, USA
SO Plant Journal, (1995) Vol. 7, No. 4, pp. 687-701.
ISSN: 0960-7412.
DT Article
LA English
ED Entered STN: 26 Jun 1995
Last Updated on STN: 26 Jun 1995
AB A system for ***insertional*** ***mutagenesis*** and chromosomal

rearrangement in Arabidopsis has been developed. The T-DNA vectors are based on the maize transposon Ds, ***lox*** sites from the Cre-***lox*** site-specific recombination system, and transcriptional fusions expressing Ac transposase or Cre recombinase. The engineered transposon is termed Dslox. Transposed Dslox insertions were created by crossing plants bearing Dslox with plants expressing Ac transposase, then simultaneously selecting for excision and reinsertion in F-2 seedlings using the herbicides chlorsulfuron and phosphonothricin, respectively. F-2 plants bearing stable Dslox insertions were identified by scoring for the absence of the Ac transposase T-DNA, using a novel, visual marker in that T-DNA. Two independent Dslox insertions were characterized and placed 5.6 and 16.5 cM from their T-DNAlox, which mapped close to m506 on chromosome 4. Plants bearing either of the two different transposed Dsloxs and T-DNA/ox were crossed to plants expressing Cre recombinase, which catalyzed recombination between the ***lox*** site in transposed Dslox and the ***lox*** site in TDNA/ox. ***Lox*** - ***lox*** recombinants were identified selectively amongst progeny of these crosses. Molecular and genetic analysis of the ***lox*** - ***lox*** rearrangements indicated that both were inversions. The smaller inversion was germinally transmitted from generation to generation as a simple trait, whereas the larger inversion was not transmitted to progeny of plants bearing the rearrangement.

=> s splice acceptor and ires and lox
L15 3 SPLICE ACCEPTOR AND IRES AND LOX

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L16 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout vector for gene trapping and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene trapping and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a ***splice*** ***acceptor*** sequence; (b) an internal ribosomal entry site (***IRES***) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 ***splice*** ***acceptor*** and polyadenylation signal, and human gene GTX element ***IRES*** . The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L16 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene trap retroviral vector pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the ***splice*** ***acceptor*** in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x- ***IRES*** -DR-bGHpA (S/A: branch site and ***splice*** ***acceptor*** from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the ***splice*** ***acceptor*** that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream ***IRES*** enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L16 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the

libraries. An exemplary vector pDKO2 designed to trap transcriptionally active genes using the function of the ***splice*** ***acceptor*** in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x- ***IRES*** -DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and ***splice*** ***acceptor*** (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the ***splice*** ***acceptor*** provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. ***IRES*** enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

=> s splice acceptor
L17 2872 SPLICE ACCEPTOR

=> d his

(FILE 'HOME' ENTERED AT 19:09:44 ON 07 AUG 2007)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:09:57 ON 07 AUG 2007
L1 696 S TRAP? (3A) VECTOR
L2 7 S L1 AND IRES AND LOX
L3 6 DUP REM L2 (1 DUPLICATE REMOVED)
L4 59 S L1 AND SPLICE ACCEPTOR
L5 4 S L4 AND LOX
L6 4 DUP REM L5 (0 DUPLICATES REMOVED)
L7 14 S L1 AND LOX
L8 11 DUP REM L7 (3 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 19:17:21 ON 07 AUG 2007.

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:18:33 ON 07 AUG 2007
L9 4930 S TRAP? AND VECTOR
L10 20 S L9 AND LOX
L11 15 DUP REM L10 (5 DUPLICATES REMOVED)
L12 21182 S INSERTION? (3A)(MUTATION OR MUTAGEN?)
L13 30 S L12 AND LOX
L14 23 DUP REM L13 (7 DUPLICATES REMOVED)
L15 3 S SPLICE ACCEPTOR AND IRES AND LOX
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)
L17 2872 S SPLICE ACCEPTOR

=> s l12 or l9
L18 25991 L12 OR L9

=> s l18 and l17
L19 146 L18 AND L17

=> s l19 and ires
L20 19 L19 AND IRES

=> dup rem l20
PROCESSING COMPLETED FOR L20
L21 19 DUP REM L20 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 19 ANSWERS - CONTINUE? Y/(N):y

L21 ANSWER 1 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:510742 CAPLUS <<LOGINID::20070807>>
DN 145:2128
TI Gene ***trap*** cassettes for random and targeted conditional gene inactivation
IN Von Melchner, Harald; Schnuetgen, Frank; Wurst, Wolfgang; Ruiz, Patricia; De-Zolt, Silke; Floss, Thomas; Hansen, Jens
PA Frankgen Biotechnologie A.-G., Germany; GSF Forschungszentrum fuer Umwelt
und Gesundheit G.m.b.H.; MPG Max-Planck-Gesellschaft zur Foerderung der Wissenschaften e.V.
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006056617	A1	20060601	WO 2005-EP56282	20051128
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 1662005	A1	20060531	EP 2004-28194	20041126
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR, IS, YU				
EP 1715053	A1	20061025	EP 2005-103092	20050418
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
PRAI EP 2004-28194	A	20041126		
EP 2005-103092	A	20050418		

AB The present invention provides for a new type of gene ***trap*** cassettes, which can induce conditional mutations. The cassettes rely on directional site-specific recombination systems, which can repair and re-induce gene ***trap*** mutations when activated in succession. After the gene ***trap*** cassettes are inserted into the genome of the target organism, mutations can be activated at a particular time and place in somatic cells. Moreover, the invention relates to the use of said cell for identification and/or isolation of genes and for the creation of transgenic organisms to study gene function at various developmental stages, including the adult, as well as for the creation of animal models of human disease useful for in vivo drug target validation. In conclusion, the present invention provides a process which enables a temporally and/or spatially restricted inactivation of all genes that constitute a living organism. Two gene ***trap*** vectors were designed for large scale conditional mutagenesis in ES cells. The first ***vector*** FlipRosa.beta.geo contains a classic ***splice*** ***acceptor*** (SA) - .beta.-galactosidase/neomycintransferase fusion gene (.beta.geo) - polyadenylation sequence (pA) cassette inserted into the backbone of a promoter- and enhancerless Moloney murine leukemia virus in inverse transcriptional orientation relative to the virus [Friedrich, G. & Soriano, P. (1991) Genes Dev. 5, 1513-1523]. The second ***vector*** FlipRosaCeo is similar to FlipRosa.beta.geo except that SA.beta.geo has been exchanged with Ceo, which is an in frame fusion between the human CD2 cell surface receptor- and the neomycin resistance genes (Gebauer, M. et al., Genome Res 11, 1871-7 (2001)). Unlike .beta.geo, Ceo does not require an extra ***splice*** ***acceptor*** site for ***trapping*** as it contains a powerful cryptic 5' splice site close to its 5' end. The mechanism relies on two site-specific recombination systems (FLPe/frt; Cre/loxP), which enable gene ***trap*** cassette inversions from the sense, coding strand of a ***trapped*** gene to the antisense, noncoding strand and back. As a result, the gene ***trap*** vectors allow (i) high throughput selection of gene ***trap*** lines using G418, [ii] inactivation of gene ***trap*** mutations prior to ES cell line conversion into mice by blastocyst injection, and (iii) reactivation of the mutations at prespecified times and in selected tissues of the resulting mice.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 2 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:103582 CAPLUS <<LOGINID::20070807>>
DN 144:165272
TI Genetic engineering mammalian genomes by integrating specific vectors and screening for cells comprising the ***vector*** inserted into the gene of interest
IN Finney, Robert E.
PA USA
SO U.S. Pat. Appl. Publ., 26 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2006024819	A1	20060202	US 2004-903001	20040730
PRAI US 2004-903001		20040730		

AB The invention relates to genetically engineering mammalian genomes by integrating specific vectors followed by screening method that allows to select cells comprising the ***vector*** inserted into the gene of interest. The invention relates to integration vectors for modifying a target genomic region comprising, in a 5' to 3' direction, a ***splice*** ***acceptor*** site, a 3' hybrid recognition site, and a marker sequence (i.e., a 5' gene ***trap*** ***vector***); or alternatively comprising, in a 5' to 3' direction, a marker sequence; a 5' hybrid recognition site; and a splice donor site (i.e., a 3' gene ***trap*** ***vector***). The integration ***vector*** , upon insertion into the target genomic region is capable of producing a recombinant RNA transcript that is comprised of a hybrid recognition site for a selection mol. The hybrid recognition site of recombinant RNA produced from insertion of the 5' gene ***trap*** ***vector*** is comprised of a 5' hybrid recognition site derived from genomic sequence and a 3' hybrid recognition site derived from ***vector*** sequence. The selection mol. selects recombinant cells comprising the integration ***vector*** inserted within the target genomic region.

L21 ANSWER 3 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout ***vector*** for gene ***trapping*** and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene ***trapping*** and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a ***splice*** ***acceptor*** sequence; (b) an internal ribosomal entry site (***IRES***) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 ***splice*** ***acceptor*** and polyadenylation signal, and human gene GTX element ***IRES*** . The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L21 ANSWER 4 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene ***trap*** vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one ***mutagen*** is an ***insertional*** ***mutagen*** , to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by ***insertional*** ***mutagenesis*** is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene ***trap*** retroviral ***vector*** pDKO2 (also called ***insertional*** ***mutagen***) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to ***trap*** transcriptional active genes using the function of the ***splice*** ***acceptor*** in the ***vector*** and the Lox signals-flanked gene ***trap*** portion contains S/A-x- ***IRES*** -DR-bGHpA (S/A: branch site and ***splice*** ***acceptor*** from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the ***vector*** is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the ***trapped*** endogenous gene onto the ***splice*** ***acceptor*** that is provided by the ***vector*** . Once this splicing event occurs, a fusion transcript will be made that results in the introduction of ***vector*** encoded stop codons that cause the premature termination of the protein product of the ***trapped*** gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream ***IRES*** enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene ***trap*** efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L21 ANSWER 5 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:894602 CAPLUS <<LOGINID::20070807>>
DN 141:375508
TI Gene ***trap*** vectors for gene mutagenesis and gene discovery
IN Zambrowicz, Brian; Friedrich, Glenn A.; Lilleberg, Stan; Sands, Arthur T.
PA Lexicon Genetics Incorporated, USA
SO U.S., 33 pp., Cont.-in-part of U.S. Ser. No. 276,533.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6808921	B1	20041026	US 1999-443282	19991119
US 6436707	B1	20020820	US 1999-276533	19990325
EP 1584689	A1	20051012	EP 2005-14225	19991119
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
US 2002081668	A1	20020627	US 2000-728446	20001130
US 2002182724	A1	20021205	US 2002-158735	20020529
US 6776988	B2	20040817		
US 2004259253	A1	20041223	US 2004-797613	20040309
US 2005095713	A1	20050505	US 2004-916782	20040811
PRAI US 1998-79729P	P	19980327		
US 1998-81727P	P	19980414		
US 1998-109302P	P	19981120		
US 1999-276533	A2	19990325		
EP 1999-960476	A3	19991119		
US 1999-443282	A1	19991119		
US 1999-168270P	P	19991201		
US 2002-158735	A1	20020529		

AB Novel vectors are described that incorporate, inter alia, a novel 3' gene ***trap*** cassette that does not encode a marker conferring antibiotic resistance and which can be used to efficiently ***trap*** , mutagenize, and identify previously unknown cellular genes. The presently described 3' gene ***trap*** cassette comprises in operable combination: a promoter region, an exon (typically characterized by a translation initiation codon and open reading frame and/or internal ribosome entry site), a splice donor sequence, and optionally, intronic sequences. The splice donor sequence is operatively positioned such that the exon of the 3' gene ***trap*** cassette is spliced to the ***splice*** ***acceptor*** site of a downstream exon or a cellularly encoded exon. The vectors typically allow several-fold to more than an order of magnitude greater no. of genes to be ***trapped*** and identified by exon sequence as compared to initial 3' gene ***trap*** vectors that utilize an exon encoding a selectable marker activity. The vectors can also incorporate 3' and/or 5' gene ***trap*** cassettes that are engineered to increase the probability of identifying the 5' ends of the open reading frames of genes. The 5' gene ***trap*** cassette comprises a selectable marker gene preceded by a ***splice*** ***acceptor*** sequence followed by a polyadenylation sequence. Mutagenesis enhancer cassettes such as a unidirectional transcription termination sequence, a mutagenic terminal exon, and a self-cleaving RNA coding region may also be included. Vectors incorporating the presently described 3' gene ***trap*** cassette find particular application in gene discovery, and the prodn. of mutated cells and animals.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 6 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral ***vector*** ***insertional*** ***mutagenesis***
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,

FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2002343616 A1 20030519 AU 2002-343616 20021104
US 2003134421 A1 20030717 US 2002-288555 20021104
EP 1451295 A2 20040901 EP 2002-780573 20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
PRAI US 2001-330978P P 20011102
WO 2002-US35405 W 20021104
AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as ***insertional*** ***mutagens*** . Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the ***insertional*** ***mutagen*** (s). The ***insertional*** ***mutagen*** thus allows identification of one or more genes that are mutated by ***insertion*** of an ***insertional*** ***mutagen*** . The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an ***insertional*** ***mutagen*** incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the ***insertional*** ***mutagens*** . The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary ***vector*** pDKO2 designed to ***trap*** transcriptionally active genes using the function of the ***splice*** ***acceptor*** in the ***vector*** is described. PDKO2 (3'LTR-lox-S/A-x- ***IRES*** -DR-bGHpA-TK-PGK-lox-Y-5'LTR) contains ***vector*** backbone from self-inactivating retroviral ***vector*** pSIR and genetic elements including S/A:branch site and ***splice*** ***acceptor*** (from the intron of an immunoglobin gene heavy chain variable region), lox:lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and PHI.:retrovirus packaging signal. Theor., when the ***vector*** is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the ***splice*** ***acceptor*** provided by the ***vector*** . Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the ***trapped*** gene. ***IRES*** enables the expression of the drug selection marker when an active promoter is ***trapped*** , which allows selection of gene ***trap*** event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene ***trapping*** is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L21 ANSWER 7 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:335255 CAPLUS <<LOGINID::20070807>>
DN 138:332879
TI ***Insertional*** and physicochemical ***mutagenesis*** methods for making homozygous gene mutations in cell lines and animals
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 165 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003035834	A2	20030501	WO 2002-US33714	20021022
WO 2003035834	A3	20030807		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002342087	A1	20030506	AU 2002-342087	20021022
EP 1446482	A2	20040818	EP 2002-776253	20021022
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2005532782	T	20051104	JP 2003-538335	20021022

PRAI US 2001-336497P P 20011022
US 2002-196721 A 20020715
WO 2002-US33714 W 20021022
AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one ***mutagen*** is an ***insertional*** ***mutagen*** , to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by ***insertional*** ***mutagenesis*** is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by combined mutagenesis of Jurkat cells in a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis.

L21 ANSWER 8 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:950663 CAPLUS <<LOGINID::20070807>>
DN 140:19765
TI Compns. and methods for making and detecting gene mutations in transgenic cell lines and animals
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 61 pp., Cont.-in-part of U.S. Ser. No. 277,612.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004018624	A1	20040129	US 2002-277612	20021022

PRAI US 2001-336497P P 20011022
US 2002-196721 B2 20020715
US 2002-277612 A2 20021022
AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms by ***insertional*** ***mutagenesis*** . The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one ***mutagen*** is an ***insertional*** ***mutagen*** , to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype, to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by ***insertional*** ***mutagenesis*** is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The examples show the use of these methods in mutagenesis of embryonic stem cells, and in the identification of gene knockouts that cause resistance to FasL-mediated apoptosis in Jurkat cells using a gene ***trap*** retroviral ***vector*** as an ***insertional*** ***mutagen*** /tag.

L21 ANSWER 9 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:633346 CAPLUS <<LOGINID::20070807>>
DN 139:174840
TI Gene ***trap*** vectors using the Sleeping Beauty transposition mechanism for integration into a target genome and their use in functional genomics
IN Hackett, Perry B.; Clark, Karl J.; Ivics, Zoltan; Izsvak, Zsuzsanna; Fahrenkrug, Scott C.
PA Regents of the University of Minnesota, USA
SO U.S. Pat. Appl. Publ., 72 pp., Cont. of U. S. Ser. No. 191,572, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003154500	A1	20030814	US 2002-191698	20020709
US 7160682	B2	20070109		

PRAI US 1998-191572 B1 19981113
AB Dicistronic gene ***trap*** vectors that use the Tc1 family of Sleeping Beauty transposons of teleost fish to integrate the ***vector*** into a host genome are described. The vectors carry a selectable or scorable marker and a weak promoter from which a gene will be expressed upon integration of the ***vector*** . These vectors may contain elements that will improve expression of a gene, such as ***splice*** ***acceptor*** sites and the promoter will be downstream of an internal ribosome entry site. Construction of the vectors is described and methods of using them in functional genomics, including mapping mutagenesis and gene discovery are discussed.

L21 ANSWER 10 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:590700 CAPLUS <<LOGINID::20070807>>
DN 139:129089
TI Methods for making polynucleotide libraries, polynucleotide arrays, and cell libraries for high-throughput genomics analysis
IN Finney, Robert E.; Lofquist, Alan

PA USA
SO U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 172,715.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003143597	A1	20030731	US 2002-291235	20021108
US 2002094536	A1	20020718	US 2001-28970	20011228
US 2002150945	A1	20021017	US 2002-172715	20020613
US 2004137490	A1	20040715	US 2003-741084	20031220
PRAI US 2000-258388P	P	20001228		
US 2001-28970	A2	20011228		
US 2002-383782P	P	20020530		
US 2002-172715	A2	20020613		
US 2002-97431	A2	20020315		
US 2002-291235	A1	20021108		

AB A method for high-throughput genomic anal. that can be used to identify the therapeutic or diagnostic utility of genes that uses a library of cells with targeted gene disruptions is described. These cells can be used to monitor phenotypes in a no. of contexts, such as testing drug candidates. Arrays of such cells can be used to monitor such disrupted cells phenotypically in the context, for example, of testing drug candidates. Knockout vectors for use in gene inactivation and that can be recovered by virtue of an origin of replication or a host cell selection marker sequence are described. The recovered sequences can be used to identify the disrupted genes or to make homologous recombination vectors, which in turn can be employed to make multi-allele knockout cells. Double-stranded RNA mols. designed to target the recovered polynucleotide are used to down regulate the polynucleotide in vitro and in vivo, following detn. of a therapeutically effective dosage of the RNAi mol. Polynucleotides that comprise part of the disrupted genes can be recovered from such "knockout" cells, by virtue of an origin of replication or a host cell selection marker sequence that is part of the construct.

L21 ANSWER 11 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:5537 CAPLUS <<LOGINID::20070807>>
DN 138:50825
TI Induction gene ***trap*** and reporter gene methods for analysis of intracellular regulatory pathways in animal cells and analysis of disease states
IN Khodadoust, Mehran M.
PA USA
SO U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S. Ser. No. 908,305.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003003519	A1	20030102	US 2001-29471	20011025
US 2002076688	A1	20020620	US 2001-908305	20010717
CA 2426896	A1	20020502	CA 2001-2426896	20011026
JP 2004528815	T	20040924	JP 2002-537853	20011026
NZ 525480	A	20060630	NZ 2001-525480	20011026
NO 2003001847	A	20030626	NO 2003-1847	20030424
PRAI US 2000-697843	B2	20001027		
US 2001-908305	A2	20010717		
US 2001-29471	A	20011025		
WO 2001-US47855	W	20011026		

AB Methods and vectors are disclosed for discovering intracellular regulatory pathways utilized by specific stimulatory agents. Suitable stimulatory agents include cytokines, chem. agents, and antibodies. Cell lines and regulatory factors are provided for screening libraries of drug candidates to identify potential therapeutic agents. Methods and compns. are also provided for identifying genes which are necessary for, or capable of, up regulating or down regulating the targeted genomic loci in the selected cells. The basic ***vector*** is an integrating ***vector*** that includes a promoterless reporter gene and selectable marker that are translated from a polycistronic transcript because of the presence of internal ribosome entry sites upstream of the genes. The ***vector*** is introduced into cells and they are exposed to an inducer of interest and a selection agent. The selection agent can be a pos. or neg. selection agent depending upon the marker gene and selection system used, e.g. ganciclovir selection can be used with a thymidine kinase marker to select against constitutive promoters. Cells that survive selection can then be tested for expression of the reporter gene.

L21 ANSWER 12 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:674660 CAPLUS <<LOGINID::20070807>>
DN 137:196652
TI Methods for making polynucleotide libraries, polynucleotide arrays, and cell libraries for high-throughput genomics analysis
IN Lofquist, Alan; Finney, Robert E.; Leung, David
PA Pangenex, Inc., USA
SO U.S. Pat. Appl. Publ., 32 pp., Cont.-in-part of U. S. Ser. No. 28,970.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002123065	A1	20020905	US 2002-97431	20020315
US 2002094536	A1	20020718	US 2001-28970	20011228
US 2002150945	A1	20021017	US 2002-172715	20020613
US 2004137490	A1	20040715	US 2003-741084	20031220
PRAI US 2000-258388P	P	20001228		
US 2001-28970	A2	20011228		
US 2002-97431	A2	20020315		
US 2002-383782P	P	20020530		
US 2002-172715	A2	20020613		
US 2002-291235	A1	20021108		

AB A method for high-throughput genomics anal., to identify the therapeutic or diagnostic utility of genes, entails the use of a construct to disrupt a gene or alleles of a gene in cells of interest. Arrays of such cells can be used to monitor such disrupted cells phenotypically in the context, for example, of testing drug candidates. Polynucleotides that comprise part of the disrupted genes can be recovered from such "knockout" cells, by virtue of an origin of replication or a host cell selection marker sequence that is part of the construct. The recovered polynucleotides can be used to identify the disrupted genes or to make homologous recombination vectors, which in turn can be employed to make multi-allele knockout cells.

L21 ANSWER 13 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:545855 CAPLUS <<LOGINID::20070807>>
DN 135:133112
TI Identification of modulator regulated genes by using vectors comprising a survival gene linked to a ***splice*** - ***acceptor*** and an internal ribosome entry site sequence
IN Lofquist, Alan; Leung, David
PA Cell Therapeutics, Inc., USA
SO PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001053481	A1	20010726	WO 2001-US1480	20010118
W: JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
PRAI US 2000-177250P	P	20000120		

AB The present invention relates to methods and compns. for the identification of portions of the genome which are modulated by compds. The invention also relates to a plurality of eukaryotic cells having integrated at a plurality of integration sites the nucleic acids of the present invention. The invention uses a set of gene ***trap*** vectors to screen and to identify modulator regulated genes. These vectors are base on a survival gene with both dominant pos. and neg. selection properties placed downstream from a ***splice*** - ***acceptor*** and an internal ribosome entry site sequence (***IRES***). The vectors allows ***trapping*** of regulated promoters when integration occurs downstream of a promoter of interest as well as within an intron or exon sequence.
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 14 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:300862 CAPLUS <<LOGINID::20070807>>
DN 134:321557
TI Conditional gene ***trapping*** construct for mutational inactivation of all genes in mammalian cells
IN Kuehn, Ralf; Von Melchener, Harald; Altschmied, Joachim
PA Artemis Pharmaceuticals Gmbh, Germany; Frankgen Biotechnologie Ag
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001029208	A1	20010426	WO 2000-EP10162	20001016
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1092768	A1	20010418	EP 1999-120592	19991016
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CA 2387737	A1	20010426	CA 2000-2387737	20001016
EP 1222262	A1	20020717	EP 2000-974397	20001016
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003512053	T	20030402	JP 2001-532191	20001016
AU 782960	B2	20050915	AU 2001-12723	20001016
PRAI EP 1999-120592	A	19991016		
US 1999-162016P	P	19991027		

WO 2000-EP10162 W 20001016
AB The present invention relates to a gene ***trapping*** construct which causes conditional mutations in genes, and the use of this gene ***trapping*** construct to mutationally inactivate all cellular genes. The gene ***trapping*** constructs comprises a functional DNA segments inserted in sense or antisense direction relative to the transcriptional orientation of the gene to be ***trapped*** and being flanked by two recombinase recognition sequences RRSs which are specific to site specific recombinase capable of inverting double stranded DNA segment. In addn. the invention relates to a cell, preferably a mammalian cell which contains the above mentioned construct. Moreover, the invention relates to the use of said cell for identification and/or isolation of genes and for the creation of transgenic organisms to study gene function at various developmental stages, including the adult. In conclusion, the present invention provides a process which enables a temporally and/or spatially restricted inactivation of all genes that constitute a living organism.
RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 15 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:742299 CAPLUS <<LOGINID::20070807>>
DN 133:306327
TI Polycistronic reporter constructs and host cells for promoter ***trapping*** and the characterization of the activities of biological response modifiers
IN Xu, Hua; Jamigan, Kurt; Zhou, Hua; Greene, Amy; Thode, Silke
PA Iconix Pharmaceuticals Inc., USA
SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000061809	A2	20001019	WO 2000-US9267	20000407
WO 2000061809	A3	20020221		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-128631P P 19990409
AB Panels of expression constructs using reporter genes under the control of different promoters and host cell lines are described for use in the characterization of effectors of biol. responses. The methods and ***vector*** combinations allow the rapid and parallel establishment of assays that reflect the activity of biol.-response-modifiers, including expressed heterologous cDNA or genes. The assays are directly utilizable as screening assays for compds. that modulate the activities of the biol.-response-modifiers. The vectors have three major components, the reporter gene cassette, a pos. selection marker and a neg. selection marker that are under control of the same promoter with an ***IRES*** used to allow translation of all three coding regions on the transcript.

L21 ANSWER 16 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:291223 CAPLUS <<LOGINID::20070807>>
DN 132:318598
TI Gene ***trap*** vectors for neuronal specific expression in vertebrates
IN Tessier-lavigne, Marc; Skarnes, William C.; Mitchell, Kevin; Leighton, Philip A.
PA The Regents of the University of California, USA
SO PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000024881	A1	20000504	WO 1999-US25105	19991026

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6248934 B1 20010619 US 1998-191652 19981113
AU 2000013238 A 20000515 AU 2000-13238 19991026
PRAI US 1998-105602P P 19981026
US 1998-191652 A 19981113
WO 1999-US25105 W 19991026
AB The invention provides methods and compns. for expressing targeted gene products in vertebrate neurons. The compns. include gene ***trap*** vectors comprising a polynucleotide comprising promoterless selectable marker and axon reporter encoding sequences, which may be operatively joined to an internal ribosome-entry site, and may comprise a ***splice*** ***acceptor*** side located 5' to the selectable marker and axon reporter encoding sequences. The methods include methods of expressing an axon reporter in a cell by transferring the subject vectors into an embryonic stem cell and incubating the cell under conditions whereby the cell or a progeny of the cell differentiates into a neuron comprising an axon or dendrites, and the neuron expressed the axon reporter under the transcriptional control of the gene; and specifically detecting the axon reporter in the axon or dendrites. Neuronal specific expression may also be effected in disclosed binary systems.
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 17 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:640997 CAPLUS <<LOGINID::20070807>>
DN 131:282372
TI Vectors containing 3' gene ***trap*** cassettes for gene mutagenesis and gene discovery
IN Zambrowicz, Brian; Friedrich, Glenn A.; Sands, Arthur T.
PA Lexicon Genetics Incorporated, USA
SO PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9950426	A1	19991007	WO 1999-US6474	19990326

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6080576 A 20000627 US 1998-57328 19980408
CA 2323834 A1 19991007 CA 1999-2323834 19990326
AU 9932036 A 19991018 AU 1999-32036 19990326
AU 751520 B2 20020815
EP 1066392 A1 20010110 EP 1999-914126 19990326
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2002509727 T 20020402 JP 2000-541314 19990326
JP 3725782 B2 20051214
US 2005095713 A1 20050505 US 2004-916782 20040811
PRAI US 1998-79729P P 19980327
US 1998-57328 A 19980408
US 1998-81727P P 19980414
US 1998-109302P P 19981120
US 1999-276533 A1 19990325
WO 1999-US6474 W 19990326
US 2002-158735 A1 20020529
AB Novel vectors are described that incorporate, inter alia , a novel 3' gene ***trap*** cassette which can be used to efficiently ***trap*** and identify previously unknown cellular genes. Efficient methods of 3' gene ***trapping*** are provided that allow a greater percentage of genes in the target cell genome to be ***trapped*** and rapidly identified. The presently described 3' gene ***trap*** cassette comprises in operable combination, a promoter region, an exon (typically characterized by a translation initiation codon and open reading frame and/or internal ribosome entry site), a splice donor sequence, and, optionally, intronic sequences. The splice donor sequence is operatively positioned such that the exon of the 3' gene ***trap*** cassette is spliced to the ***splice*** ***acceptor*** site of a downstream exon or a cellularly encoded exon. In a preferred embodiment, the exon component of the 3' gene ***trap*** cassette, which also serves as a sequence acquisition cassette, will comprise exon sequence and a splice donor sequence derived from genetic material that naturally occurs in an eukaryotic cell. Addnl. embodiments of the present invention include recombinant vectors, particularly viral vectors, that have been genetically engineered to incorporate the 3' gene ***trap*** cassette. The vectors can also be engineered to include a 5' gene ***trap*** cassette that typically contains a ***splice*** ***acceptor*** site located 5' to an exon (which can encode a selectable marker gene) followed by an operatively positioned polyadenylation sequence. The splicing machinery is better able to recognize an exon type sequence present adjacent to or relatively close to a promoter when splicing into downstream exons. Vectors incorporating the described 3' gene ***trap*** cassette find particular application in gene discovery and in the prodn. of mutated cells and animals.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 18 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:64965 CAPLUS <<LOGINID::20070807>>
DN 130:120465
TI A two-component system for identification and tagging of genes showing tissue- or cell-limited expression
IN Ong, Christopher J.; Priatel, John J.; Jirik, Frank R.
PA The University of British Columbia, Can.

SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9902719	A1	19990121	WO 1998-CA677	19980710
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2205888	A1	19990111	CA 1997-2205888	19970711
CA 2295475	A1	19990121	CA 1998-2295475	19980710
EP 1003893	A1	20000531	EP 1998-933402	19980710
R: AT, BE, CH, DE, DK, FR, GB, IT, LI, NL, SE				
US 6777235	B1	20040817	US 1999-295464	19990419
PRAI CA 1997-2205888	A	19970711		
WO 1998-CA677	W	19980710		

AB Reporter genes and methods for their use that allow the detection and cloning of eucaryotic genes whose expression is restricted to certain tissues or specialized cell types are described. The method uses two reporter genes, one is under control of a promoter selected for its restricted function in a particular cell or tissue. The second gene is a promoter-less reporter gene that may have a 5'-terminal ***IRES*** element to allow in-frame translation of the transcript. In addn., there may be a ***splice*** ***acceptor*** site upstream of the second gene to ensure that it is not lost during post-transcriptional processing. The two genes are introduced into target ells using independent vectors or transformations. Transformed cells are allowed to differentiate to produce a specialized cell or tissue that is monitored for expression of both reporter genes, thereby detecting a gene into which the ***trap*** ***vector*** has integrated which is expressed in the same cell or tissue type as the selected promoter. Preferably, the two reporter gene products are subunits of a single protein that do not have sep. functions, e.g. the .alpha.-complementing and .omega. peptides of .beta.-galactosidase. Alternatively, two-hybrid systems can be used with expression of both components necessary or expression of a reporter gene. A system depending upon the interaction of aequorin and green fluorescent protein is also described.
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 19 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:439294 CAPLUS <<LOGINID::20070807>>
DN 131:69280
TI Novel gene ***trap*** and its use for high efficiency selection of regulated eukaryotic genes
IN Baetscher, Manfred; Nir, Waan-jeng
PA Biotransplant, Inc., USA
SO U.S., 23 pp., Cont. of U.S. Ser. No. 374,833, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5922601	A	19990713	US 1996-716854	19960916
PRAI US 1995-374833	B1	19950119		

AB The invention provides a novel gene ***trap*** construct that allows for high efficiency identification and selection of eukaryotic genes whose activity is regulated upon a cellular transition. Said viral ***vector*** comprises in its downstream sequence (i) a cassette having a functional ***splice*** ***acceptor*** , a translation stop sequence and an internal ribosome entry site and (ii) a promoterless protein coding sequence encoding at least one polypeptide providing pos. and neg. selection traits. Also provided is a method for identification of genes whose activity is regulated upon a cellular transition event by introducing the gene ***trap*** construct into a cell and observing expression of the pos. and/or neg. selection traits before and after the transition event.
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s ires and lox
L22 18 IRES AND LOX

=> dup rem l22
PROCESSING COMPLETED FOR L22
L23 15 DUP REM L22 (3 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 15 ANSWERS - CONTINUE? Y/(N):y

L23 ANSWER 1 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:346491 CAPLUS <<LOGINID::20070807>>
DN 145:118305
TI Preparation mammalian containing gene targeting site for expressing medically useful proteins in mammary gland
IN Yuan, Sanping; Zhang, Yijing; Xian, Jian
PA Qingdao Senmiao Biotechnology Institute, Peop. Rep. China
SO Faming Zhuanli Shenqing Gongkai Shuomingshu, 25 pp.
CODEN: CNXXEV
DT Patent
LA Chinese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI CN 1730659	A	20060208	CN 2005-10055256	20050317
PRAI CN 2005-10055256		20050317		

AB This invention provides a process of prepn. of mammallian contg. gene targeting site ***Lox*** . The title method comprises (1) constructing a gene targeting vector by incorporating a gene encoding medicinal protein, a ***Lox*** sequence, a poly-A signal, a selective marker gene and another ***Lox*** sequence in the expression cassette of mammalian milk protein gene; (2) transforming a host cell and screening for transformants; (3) detg. gene-targeted cell strains by PCR and Southern blot; (4) constructing cloned animal by transplantation of the nucleus of gene-targeted cell strains; (5) propagating the cloned animal; (6) introducing Cre gene into fertilized ovum or somatic cell of the gene-targeted animal to obtain a new gene-targeted animal without the selective marker gene; and (7) using Cre- ***lox*** and ***IRES*** to introduce another medicinal protein-encoding gene into the gene-targeted animal to construct a bioreactor capable of producing second medicinal proteins. The aminal provided in this invention can be used for expressing medically useful proteins in mammary gland.

L23 ANSWER 2 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:530217 CAPLUS <<LOGINID::20070807>>
DN 144:1239
TI Characterization of an exchangeable gene trap using pU-17 carrying a stop codon-.beta.geo cassette
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi; Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, 862-0976, Japan
SO Development, Growth & Differentiation (2005), 47(3), 163-172
CODEN: DGDFA5; ISSN: 0012-1592
PB Blackwell Publishing Asia Pty Ltd.
DT Journal
LA English
AB We have developed anew exchangeable gene trap vector, pU-17, carrying the intron-lox71-splicing acceptor (SA)-.beta.-geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of .beta.galactosidase/neomycin-resistance fusion gene (.beta.geo) that can function in promoter trapping. We found that the trap vector was highly selective for integrations in the introns adjacent to the exon contg. the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the .beta.geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the .beta.geo gene. Thus, using this pU-17 trap vector, we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the .beta.geo gene with any gene of interest to be expressed under the control of the trapped promoter through Cre-mediated recombination.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:718658 CAPLUS <<LOGINID::20070807>>
DN 141:205054
TI Methods for generating PTEN-deficient cells via somatic cell gene targeting and their use in monitoring cancer pathogenesis and screening for anti-cancer agents
IN Waldman, Todd; Lee, Carolyn
PA Georgetown University, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004074459	A2	20040902	WO 2004-US5179	20040219
WO 2004074459	A3	20060526		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
PRAI US 2003-448799P	P	20030219		

AB The present invention provides isogenic pairs of human cells that differ only in their PTEN alleles. In particular, it relates to methods for generating PTEN-deficient cells via somatic cell gene targeting and their

use in monitoring cancer pathogenesis and screening for anti-cancer agents.

L23 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:292111 CAPLUS <<LOGINID::20070807>>
DN 140:298627
TI Efficient generation of stable expression cell lines through the use of scorable homeostatic reporter genes for production of protein complexes
IN Dubridge, Robert B.
PA Protein Design Labs, Inc., USA
SO PCT Int. Appl., 90 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004029284	A2	20040408	WO 2003-US31311	20030930
WO 2004029284	A3	20050512		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003283995	A1	20040419	AU 2003-283995	20030930
US 2004115814	A1	20040617	US 2003-676476	20030930
US 2006286671	A1	20061221	US 2006-509177	20060823
PRAI US 2002-415216P	P	20020930		
US 2003-676476	A1	20030930		
WO 2003-US31311	W	20030930		

AB The present invention provides compns., systems and methods for identifying and utilizing advantageous genomic sites for expression of recombinant proteins. This is accomplished by randomly inserting plastic expression systems that permit exchange of their coding regions while leaving the remainder of the expression system, including the promoter, in place. More specifically, the invention described herein provides integration cassettes that are inserted into cellular genetic material by a non-homologous recombination event. These integration cassettes comprise expression systems for selectable and scorable reporter genes that allow cells successfully transformed with the integration cassettes to be identified and the level of expression supported by the cassette at its site of insertion to be established. The methods and vectors for site-specific recombination in a cell of the present invention can be used to obtain persistent gene expression in a cell and to modulate gene expression. One preferred method according to the invention comprises contacting a cell with a vector comprising an origin of replication functional in mammalian cells located between first and second recombining sites located in parallel. Another preferred method comprises, in part, contacting a cell with a vector comprising first and second recombining sites in antiparallel orientations such that the vector is internalized by the cell. In both methods, the cell is further provided with a site-specific recombinase that effects recombination between the first and second recombining sites of the vector. The transformation of CHO cells using CJA8 exchangeable reporter element and scorable reporter gene, CD4, was performed. The exchanging a reporter segment for a target segment using Flp recombination system was demonstrated. The construction of an antibody library was demonstrated.

L23 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout vector for gene trapping and gene targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530
PRAI US 2003-448395	A	20030530		

AB The invention relates to a method for conditionally knocking out and altering gene function for use in gene trapping and gene targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a splice acceptor sequence; (b) an internal ribosomal entry site (***IRES***) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 splice acceptor and polyadenylation signal, and human gene GTX element ***IRES*** . The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L23 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional gene trap vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022
US 2003224519	A1	20031204	US 2003-345115	20030115
US 2004253727	A1	20041216	US 2003-342761	20030115
US 2004253589	A1	20041216	US 2003-342896	20030115
US 2004253590	A1	20041216	US 2003-342923	20030115
US 2004253591	A1	20041216	US 2003-342948	20030115
PRAI US 2001-336497P	P	20011022		
US 2002-196721	B2	20020715		
US 2002-277612	A2	20021022		

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a gene trap retroviral vector pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the splice acceptor in the vector and the ***Lox*** signals-flanked gene trap portion contains S/A-x- ***IRES*** -DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the trapped endogenous gene onto the splice acceptor that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the trapped gene. In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream ***IRES*** enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The gene trap efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L23 ANSWER 7 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:457963 BIOSIS <<LOGINID::20070807>>
DN PREV200510252431
TI Analysis of the Notch ligand Delta-4 in developmental and pathological neovascularization.
AU Mailhos, C. [Reprint Author]
CS Eyetech Pharmaceut, Woburn, MA USA
SO IOVS, (APR 2004) Vol. 45, No. Suppl. 2, pp. U490.
Meeting Info.: Annual Meeting of the Association-for-Research-in-Vision-and-Ophthalmology. Ft Lauderdale, FL, USA. April 24 -29, 2004. Assoc Res Vis & Ophthalmol.
CODEN: IOVSDA. ISSN: 0146-0404.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 9 Nov 2005
Last Updated on STN: 9 Nov 2005
AB Purpose: Delta-Notch signaling regulates cell-fate choices in tissues during metazoan development, including the cardiovasculature. Delta4 (Dl4) is a Notch ligand expressed primarily in arterial endothelium during early mouse embryogenesis. Subsequent expressions is restricted to microvessals and levels are greatly reduced in adult tissues. However, Dl4 expression is reactivated in sitesof active blood vessel growth in the adult mouse, such as the maturing follicles of the ovary, and during pathological neovascular situations. Our current aim is to better understand Dl4 function, and its potential as a vascular target in ocular

neovascular target in ocular neovascular disease.Methods: To analyse Dl4 function in embryonic development, we have guaranteed by deletion of the first exon and insertion of an ***IRES*** -beta-gal-neo cassette by homologous recombination. Endothelial-specific knockout mice were generated by insertion of ***lox*** sites flanking the first three exons of the Delta-4 gene and deletion of these segments by breeding to mice harbouring TielCre. Dl4 expression during retinal vascular development and pathological retinal neovascularization was characterized using in situ hybridization and qRT-PCR.Results: Analysis of Dl4 null mice revealed that recovery of heterozygote animals occurred at a lower frequency than expected (25-30%), suggesting a partially penetrant haplo-insufficiency. No homozygote embryos were found as early asembryonic day 7, before any vascular patterning occurs, suggesting an earlierrole for Dl4 in mouse development. Deletion of Dl4 function specifically within the developing endothelium resulted in defective aorta formation, loss of arterial identity, impaired microvessel sprouting into the neural tube and abnormal yolk sac microvasculature. Lastly, a potential role for Dl4 in ocular angiogenesis is suggested by its restricted expression at the very front of growing microvessels as they extend peripherally during retinal development, and byits upregulation during pathological retinal neovascularization.Conclusion: Dl4's vessel-specific expression, functional role in vessel morphogenesis and re-activation during pathological ocular neovascularization make it an ' attractive new target for anti-angiogenic therapy.

L23 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary vector pDKO2 designed to trap transcriptionally active genes using the function of the splice acceptor in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x- ***IRES*** -DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the trapped gene. ***IRES*** enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of gene trap event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and gene trapping is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L23 ANSWER 9 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:154807 BIOSIS <<LOGINID::20070807>>
DN PREV200400148360
TI Expression of an ABCG2/GFP allele in murine bone marrow hematopoeitic cells.
AU Tadjali, Mehrdad [Reprint Author]; Zhou, Sheng [Reprint Author]; Sorrentino, Brian P. [Reprint Author]
CS Department of Hematology/Oncology, St. Jude Children's Research Hospital, Memphis, TN, USA
SO Blood, (November 16 2003) Vol. 102, No. 11, pp. 820a. print.
Meeting Info.: 45th Annual Meeting of the American Society of Hematology. San Diego, CA, USA. December 06-09, 2003. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004
AB In our previous studies we have shown that expression of ABCG2/BCRP, a member of ATP binding protein (ABC) transporter superfamily, was associated with the side population (SP) phenotype of hematopoietic stem cells (HSCs). In particular, the loss of ABCG2 gene expression in knock-out mice led to a significant reduction in the number of SP cells in the bone marrow. In order to prospectively determine the HSC content of ABCG2-expressing hematopoietic populations, we generated mice with a GFP reporter gene under control of the ABCG2 promoter. Because an earlier attempt to target GFP to the translational start site was unsuccessful, we targeted an ***IRES*** -GFP cassette into the 3' untranslated region (UTR) of the ABCG2 locus, and subsequently removed the neor gene by Cre-***Lox*** recombination. Flow cytometry analysis of GFP expression in the bone marrow of bcrp+/GFP mice showed no GFP expression in T and B lymphocytes, macrophages, or granulocytes, consistent with our previous mRNA data in wild type mice. Significant expression of GFP in Ter119+ erythroid cells was found, also consistent with prior mRNA data and recent reports implicating ABCG2 in heme regulation (Joncker, JW et.al. PNAS, 2002 Nov 26;99(24): 15649; Zhou, S. ASH abstract, 2003). In order to study ABCG2/GFP expression in populations known to be enriched for HSCs, we used flow cytometry to gate on Lin- cells and found that 2.5% of them expressed GFP. Further gating on Lin-, c-kit+, Sca1+ (KSL) cell showed that 20% expressed GFP. These data show that ABCG2 expression correlates with known enrichment markers for HSCs. SP analysis of bone marrow cells demonstrated that approximately 13% of the bulk SP cells expressed GFP. When SP cells in the gated KSL population were analyzed, we found that 28% expressed GFP. Because the distal tip of the SP region is known to be enriched for HSCs relative to the shoulder region, we gated on these regions and found that 64% of cells in distal portion of SP region showed GFP expression, while 12% of cells in shoulder region were GFP positive. These results further substantiate the use of ABCG2 as a marker for HSCs, and show that the SP region is heterogeneous for cells expressing ABCG2. Experiments are in progress to investigate the frequency of repopulating cells in GFP positive and negative cell populations.

L23 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:849821 CAPLUS <<LOGINID::20070807>>
DN 137:364382
TI Method for generating conditional reporters by gene trapping using Cre recombinase-mediated site-specific recombination
IN Chambon, Pierre; Ghyselinck, Norbert B.; Schnuetgen, Frank
PA Association pour le Developpement de la Recherche en Genetique Moleculaire
(ADEREGEM), Fr.
SO PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002088353	A2	20021107	WO 2002-IB2493	20020419
WO 2002088353	A3	20030320		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003159160	A1	20030821	US 2001-843150	20010427
US 7074611	B2	20060711		
AU 2002304258	A1	20021111	AU 2002-304258	20020419
EP 1383891	A2	20040128	EP 2002-733179	20020419

EP 1383891 B1 20051214
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
AT 312912 T 20051215 AT 2002-733179 20020419
US 2004244071 A1 20041202 US 2004-475962 20040413
PRAI US 2001-843150 A 20010427
WO 2002-IB2493 W 20020419
AB The invention relates to a method for the stable inversion of a DNA fragment upon recombinase-mediated rearrangements using two sets of two incompatible site-specific recombinase targeting sites (SSRTS) in the same order but in reverse orientation flanking said DNA fragment to be inverted. The invention also relates to a method for the stable inversion of said DNA fragment upon rearrangement mediated by a recombinase such as Cre recombinase. The invention also relates to a method for obtaining a transgenic cell of which at least one allele of a DNA sequence of interest is invalidated by a process of conditional deletion and the genome of which comprises a reporter gene inserted at the place of the DNA fragment deleted by said process of conditional deletion. The invention also concerns a method to generate targeting sites to perform site-specific recombination mediated cassette exchange. The corresponding vectors, host cells, and transgenic animals are claimed.

L23 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:716477 CAPLUS <<LOGINID::20070807>>
DN 137:231337
TI Reporter gene-expressing recombinant cells and transgenic animals for study of the immune response and screening for immunomodulators
IN Thiam, Kader; Fraichard, Alexandre; Lapize-Gauthey, Christine
PA Genoway, Fr.
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA French
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002072820	A1	20020919	WO 2002-FR837	20020308
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
FR 2821854	A1	20020913	FR 2001-3272	20010309
FR 2821854	B1	20040716		
CA 2440191	A1	20020919	CA 2002-2440191	20020308
AU 2002247807	A1	20020924	AU 2002-247807	20020308
EP 1368455	A1	20031210	EP 2002-716884	20020308
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004115803	A1	20040617	US 2004-471465	20040203
PRAI FR 2001-3272	A	20010309		
WO 2002-FR837	W	20020308		

AB The invention relates to a transgenic non-human animal cell expressing at least one transgene coding for at least one reporter protein, characterized in that the expression of said reporter protein is correlated to the expression of at least one protein which is naturally produced by said cell and which is specific for a type of polarization of the immune response and/or an effector function of the immune response. The invention also relates to a corresponding transgenic animal. According to the invention, the cell and the transgenic animal can be used in a method for characterizing the type of immune response, i.e., Th1 and Th2, caused by an immunogen, a pathogen, or chem. agent. Thus, a vector for creating recombinant mouse cells comprising mouse interleukin 4 gene fragment, an ***IRES*** , a gene for red fluorescent protein, a Neo-TK gene flanked by ***Lox*** sites, mouse interleukin 4 gene 3'-flank, and diphtheria toxin gene was prepd. This may be used in creation of transgenic mice and anal. of the Th2 response.
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 12 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:391889 CAPLUS <<LOGINID::20070807>>
DN 136:396975
TI Construction of recombinant vectors comprising a transcriptionally silent element for conditional gene inactivation in mammalian cells
IN Xin, Hong-Bo; Kotlikoff, Michael
PA Cornell Research Foundation, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040685	A2	20020523	WO 2001-US43916	20011116
WO 2002040685	A3	20030109		
WO 2002040685	A9	20030724		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002019841	A5	20020527	AU 2002-19841	20011116
US 2004077089	A1	20040422	US 2003-416995	20031106
PRAI US 2000-249200P	P	20001116		
WO 2001-US43916	W	20011116		

AB The present invention provides recombinant vectors and methods of using the vectors in a high-throughput genetic system to rapidly generate conditional and/or conventional knockout mutants, such as mouse, useful to identify and define mammalian gene function in vivo. The methods of the invention combine gene trapping, gene targeting, and site-specific recombination techniques. The vectors comprise a transcriptionally silent genetic element that is inserted within a gene in a target cell in a manner that retains the functionality of the gene, and which element can be manipulated to inactivate the gene when desired. The vectors of the invention may be introduced ot cells via any means including non-biol. means, e.g., electroporation, or biol. means, e.g., via infection with a viral vector such as a retroviral vector.

L23 ANSWER 13 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 1
AN 2001:102468 BIOSIS <<LOGINID::20070807>>
DN PREV200100102468
TI Lipoxygenase mRNA silencing in erythroid differentiation: The 3'UTR regulatory complex controls 60S ribosomal subunit joining.
AU Ostareck, Dirk H.; Ostareck-Lederer, Antje; Shatsky, Ivan N.; Hentze, Matthias W. [Reprint author]
CS Gene Expression Programme, European Molecular Biology Laboratory, Meyerhofstrasse 1, D-69117, Heidelberg, Germany
hentze@embl-heidelberg.de
SO Cell, (January 26, 2001) Vol. 104, No. 2, pp. 281-290. print.
CODEN: CELLB5. ISSN: 0092-8674.
DT Article
LA English
ED Entered STN: 28 Feb 2001
Last Updated on STN: 15 Feb 2002
AB 15-lipoxygenase (***LOX***) expression is translationally silenced in early erythroid precursor cells by a specific mRNA-protein complex formed between the differentiation control element in the 3' untranslated region (UTR) and hnRNPs K and E1. The 3'UTR regulatory complex prevents translation initiation by an unknown mechanism. We demonstrate that the 40S ribosomal subunit can be recruited and scan to the translation initiation codon even when the silencing complex is bound to the 3'UTR. However, the joining of the 60S ribosomal subunit at the AUG codon to form a translation competent 80S ribosome is inhibited, unless initiation is mediated by the IGR- ***IRES*** of the cricket paralysis virus. These findings identify the critical step at which ***LOX*** mRNA translation is controlled and reveal that 60S subunit joining can be specifically regulated.

L23 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:133382 CAPLUS <<LOGINID::20070807>>
DN 132:176573
TI Method of performing single shot double transgenesis (one manipulation) involving microinjection of oocytes
IN Perry, Anthony C. F.; Wakayama, Teruhiko
PA USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000008924	A1	20000224	WO 1999-US18429	19990811
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2340242	A1	20000224	CA 1999-2340242	19990811
AU 9955603	A	20000306	AU 1999-55603	19990811
EP 1111991	A1	20010704	EP 1999-942164	19990811
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9913644	A	20011120	BR 1999-13644	19990811
JP 2002524054	T	20020806	JP 2000-564438	19990811
NZ 509861	A	20040130	NZ 1999-509861	19990811
ZA 2001000877	A	20030430	ZA 2001-877	20010131
MX 2001PA01586	A	20020408	MX 2001-PA1586	20010209
US 2004088748	A1	20040506	US 2003-422054	20030423
US 2006130163	A1	20060615	US 2005-265523	20051102

PRAI US 1998-96078P P 19980811
US 1999-134251P P 19990513
US 1999-133970P P 19990513
US 1999-371648 A1 19990810
WO 1999-US18118 A 19990810
WO 1999-US18429 W 19990811
US 2001-762924 A1 20010423
US 2002-285818 B1 20021031
US 2003-422054 A1 20030423

AB The invention provides a method for generating transgenic animals and cells by the coinsertion of nucleic acid and a nucleus into an unfertilized oocyte cytoplasm using a microinjection pipet. The oocyte is immature and is arrested at the second metaphase (metII) of meiosis. The nucleus may be taken from an embryo or fetus or mammalian cell or somatic cell or gamete (oocyte or spermatozoon). The mammals may include primates, ovines, bovines, porcines, ursines, caprines, felines, canines, equines, and murines. Preferably, the coinsertion is by microinjection and more preferably by piezo-elec. actuated microinjection. Transgene (tg) expressing embryos are here produced following coinjection of unfertilized mouse oocytes with sperm heads and exogenous DNA encoding either a green fluorescent protein (GFP) or .beta.-galactosidase reporter. The authors show that sperm heads whose membranes have been disrupted by freezing or freeze-drying or detergent promote transgenesis with high efficiency. The detergent may be ionic or non-ionic. This allows transgene DNA to gain access to sub-nuclear elements, including the perinuclear matrix (in case of spermatozoa), the nuclear matrix, chromatin and genomic DNA.,. The Cre- ***lox*** system was used to monitor transgenesis where loxP sites flank a tissue-specific promoter. This involves utility of site-specific recombination and uses site-specific recombinases, single-stranded DNA binding proteins, RNA binding proteins. reverse transcriptases, topoisomerases, endonucleases and recombinases that promote homologous recombination. The microinjected oocyte may be allowed to develop into differentiated cells or stem cells; into an embryo in vitro prior to transfer into a host surrogate mother; or it may be transferred directly into a host surrogate mother. Microinjection of tg DNA in absence of sperm nucleus suggests that sperm nuclear components sustain tg DNA in recombinogenic form. Embryonic development can occur to term, such that the offspring possess transgenic modifications that may alter their characteristics (phenotype) and are, in turn, transmitted to their offspring.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 15 OF 15 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 2
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene trap using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002

AB The gene trap technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of gene trapping is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene trap strategy. Here, we have constructed a new gene trap vector, pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (***IRES***)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the trap vector. By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the trap vector is lost through integration into the genome. Thus, this method, termed exchangeable gene trapping, has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

=> s gene (3a) trap?
L24 2751 GENE (3A) TRAP?

=> s l24 and lox
L25 33 L24 AND LOX

=> s l25 and ires
L26 8 L25 AND IRES

=> dup rem l26
PROCESSING COMPLETED FOR L26
L27 7 DUP REM L26 (1 DUPLICATE REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y/(N):y

L27 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:530217 CAPLUS <<LOGINID::20070807>>
DN 144:1239
TI Characterization of an exchangeable ***gene*** ***trap*** using pU-17 carrying a stop codon-.beta.geo cassette
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi; Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University, Kumamoto, 862-0976, Japan
SO Development, Growth & Differentiation (2005), 47(3), 163-172
CODEN: DGDFA5; ISSN: 0012-1592
PB Blackwell Publishing Asia Pty Ltd.
DT Journal
LA English

AB We have developed anew exchangeable ***gene*** ***trap*** vector, pU-17, carrying the intron-lox71-splicing acceptor (SA)-.beta.-geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of .beta.galactosidase/neomycin-resistance fusion gene (.beta.geo) that can function in promoter trapping. We found that the trap vector was highly selective for integrations in the introns adjacent to the exon contg. the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the .beta.geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the .beta.geo gene. Thus, using this pU-17 trap vector, we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the .beta.geo gene with any gene of interest to be expressed under the control of the trapped promoter through Cre-mediated recombination.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L27 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1036594 CAPLUS <<LOGINID::20070807>>
DN 142:18448
TI Conditional knockout vector for ***gene*** ***trapping*** and ***gene*** targeting using an inducible gene silencer for recombinase-mediated inversion
IN Askew, G. Roger; Kanki, Kim L.
PA Wyeth, John, and Brother Ltd., USA
SO U.S. Pat. Appl. Publ., 41 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004241851	A1	20041202	US 2003-448395	20030530
CA 2428972	A1	20041130	CA 2003-2428972	20030530

PRAI US 2003-448395 A 20030530

AB The invention relates to a method for conditionally knocking out and altering gene function for use in ***gene*** ***trapping*** and ***gene*** targeting. Specifically, the genetic sequence is a inducible gene silencer comprising: (a) a splice acceptor sequence; (b) an internal ribosomal entry site (***IRES***) sequence; (c) a nucleotide sequence coding for a reporter protein; (d) a polyadenylation sequence; and (e) a pair of oppositely oriented recombination site sequences flanking element (a) through (d), which cause single cycle inversions in the presence of a suitable recombinase enzyme. The invention also provides the sequences of gene silencer, element lox71 and lox66, Simian virus 40 splice acceptor and polyadenylation signal, and human gene GTX element ***IRES*** . The invention further relates to targeting of the inducible gene silencer to intron one of the HPRT locus in mouse ES cells.

L27 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:80253 CAPLUS <<LOGINID::20070807>>
DN 140:123648
TI Compositions and methods for making mutations in cell lines and animals by physicochem. treatment and insertional ***gene*** ***trap*** vectors
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO U.S. Pat. Appl. Publ., 59 pp., Cont.-in-part of U.S. Ser. No. 196,721, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004018624	A1	20040129	US 2002-277612	20021022

US 2003224519 A1 20031204 US 2003-345115 20030115
US 2004253727 A1 20041216 US 2003-342761 20030115
US 2004253589 A1 20041216 US 2003-342896 20030115
US 2004253590 A1 20041216 US 2003-342923 20030115
US 2004253591 A1 20041216 US 2003-342948 20030115
PRAI US 2001-336497P P 20011022
US 2002-196721 B2 20020715
US 2002-277612 A2 20021022

AB The present invention is directed generally to redn. or inactivation of gene function or gene expression in cells in vitro and in multicellular organisms. The invention encompasses methods for mutating cells using a combination of mutagens, particularly wherein at least one mutagen is an insertional mutagen, to achieve homozygous gene mutation or mutation of multiple genes required cumulatively to achieve a phenotype to create knock-outs, knock-downs, and other modifications in the same cell. The invention is also directed to cells (and libraries thereof) and organisms created by the methods of the invention, including those in which at least one of the genes created by insertional mutagenesis is tagged by means of the insertion sequences thereby allowing identification of the mutated gene(s). The invention is also directed to libraries of mutated cells and their uses. The invention is also directed to methods of identifying mutations with methods of the invention, in cells (and libraries thereof) and organisms, by means of the insertional tag. The invention is exemplified by identification of gene knockouts that cause resistance to FasL-induced apoptosis in Jurkat cells using a combined physicochem. and genetic mutagenesis scheme. In particular, ENU is first used to create many mutations per cell, and then a ***gene*** ***trap*** retroviral vector pDKO2 (also called insertional mutagen) is used to knock out and tag addnl. alleles in ENU clones to create phenotype and permit identification of the mutated genes. PDKO2 is designed to trap transcriptional active genes using the function of the splice acceptor in the vector and the ***Lox*** signals-flanked ***gene*** ***trap*** portion contains S/A-x- ***IRES*** -DR-bGHpA (S/A: branch site and splice acceptor from the intron of an Ig gene heavy chain variable region, x: stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR: neomycin resistance gene, bGHpA: bovine growth hormone polyA sequence). When the vector is integrated within a gene, splicing occurs from splice donors that exist at the end of exons in the ***trapped*** endogenous ***gene*** onto the splice acceptor that is provided by the vector. Once this splicing event occurs, a fusion transcript will be made that results in the introduction of vector encoded stop codons that cause the premature termination of the protein product of the ***trapped*** ***gene*** . In a cellular screen to identify gene knockouts that cause resistance to FasL-induced apoptosis. Downstream ***IRES*** enables the reinitiation of translation on the fusion transcript expressing a selectable drug resistance protein for the selection. The ***gene*** ***trap*** efficiency of PDKO2 is assessed by RT-PCR specifically for several known genes, including DHFR, HPRT, FasR, and Casp8.

L27 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:377014 CAPLUS <<LOGINID::20070807>>
DN 138:363819
TI Methods for mutating genes in cells and animals using retroviral vector insertional mutagenesis
IN Harrington, John Joseph; Jackson, Paul David; Jiang, Li
PA Athersys, Inc., USA
SO PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003040324	A2	20030515	WO 2002-US35405	20021104
WO 2003040324	A3	20031211		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002343616	A1	20030519	AU 2002-343616	20021104
US 2003134421	A1	20030717	US 2002-288555	20021104
EP 1451295	A2	20040901	EP 2002-780573	20021104
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRAI US 2001-330978P	P	20011102		
WO 2002-US35405	W	20021104		

AB The present invention is in the fields of mol. biol., cell biol., and genetics. The invention is directed generally to mutating genes in cells in vitro and in multi-cellular organisms. The invention encompasses methods for mutating genes in cells using polynucleotides that act as insertional mutagens. Such methods are used to achieve mutation of a single gene to achieve a desired phenotype as well as mutation of multiple genes, required cumulatively to achieve a desired phenotype, in a cell or in a multi-cellular organism. The invention is also directed to methods of identifying one or more mutated genes, made by the methods of the invention, in cells and in multi-cellular organisms, by means of a tagging property provided by the insertional mutagen(s). The insertional mutagen thus allows identification of one or more genes that are mutated by insertion of an insertional mutagen. The invention is also directed to methods for correlating a phenotype with a gene by screening or selecting cells that have been mutated by an insertional mutagen incorporated into one or more genes in a cell and identifying the gene or genes causing the phenotype by means of a tagging property in one or more of the insertional mutagens. The invention is also directed to cells and multi-cellular organisms created by the methods of the invention and uses of the cells and multicellular organisms. The invention is also directed to libraries of cells created by the methods of the invention and uses of the libraries. An exemplary vector pDKO2 designed to trap transcriptionally active genes using the function of the splice acceptor in the vector is described. PDKO2 (3'LTR- ***lox*** -S/A-x- ***IRES*** -DR-bGHpA-TK-PGK- ***lox*** -Y-5'LTR) contains vector backbone from self-inactivating retroviral vector pSIR and genetic elements including S/A:branch site and splice acceptor (from the intron of an immunoglobin gene heavy chain variable region), ***lox*** :lox71/lox66 sequences, cre recombinase recognition sites, x:stop codons in all 3 reading frames, ***IRES*** : wild type internal ribosomal entry site from EMCV, DR:drug resistance gene for selection in the presence of neomycin, bGHpA: bovine growth hormone polyA sequence, TK: thymidine kinase, PGK: PGK promoter, and .PHI.:retrovirus packaging signal. Theor., when the vector is integrated into a gene, splicing can occur using endogenous splice donor at the end of exons and the splice acceptor provided by the vector. Once this splicing event occurs, a fusion transcript will be made resulting in a truncated protein of the ***trapped*** ***gene*** . ***IRES*** enables the expression of the drug selection marker when an active promoter is trapped, which allows selection of ***gene*** ***trap*** event. The retrovirus produced from RetroPack PT67 cells is used to infect Jurkat and ***gene*** ***trapping*** is detected by RT-PCR in genes for DHFR, HPRT, FasR, and Casp8 using their specific primers.

L27 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:849821 CAPLUS <<LOGINID::20070807>>
DN 137:364382
TI Method for generating conditional reporters by ***gene*** ***trapping*** using Cre recombinase-mediated site-specific recombination
IN Chambon, Pierre; Ghyselinck, Norbert B.; Schnuetgen, Frank
PA Association pour le Developpement de la Recherche en Genetique Moleculaire
(ADEREGEM), Fr.
SO PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002088353	A2	20021107	WO 2002-IB2493	20020419
WO 2002088353	A3	20030320		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003159160	A1	20030821	US 2001-843150	20010427
US 7074611	B2	20060711		
AU 2002304258	A1	20021111	AU 2002-304258	20020419
EP 1383891	A2	20040128	EP 2002-733179	20020419
EP 1383891	B1	20051214		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
AT 312912	T	20051215	AT 2002-733179	20020419
US 2004244071	A1	20041202	US 2004-475962	20040413
PRAI US 2001-843150	A	20010427		
WO 2002-IB2493	W	20020419		

AB The invention relates to a method for the stable inversion of a DNA fragment upon recombinase-mediated rearrangements using two sets of two incompatible site-specific recombinase targeting sites (SSRTS) in the same order but in reverse orientation flanking said DNA fragment to be inverted. The invention also relates to a method for the stable inversion of said DNA fragment upon rearrangement mediated by a recombinase such as Cre recombinase. The invention also relates to a method for obtaining a transgenic cell of which at least one allele of a DNA sequence of interest is invalidated by a process of conditional deletion and the genome of which comprises a reporter gene inserted at the place of the DNA fragment deleted by said process of conditional deletion. The invention also concerns a method to generate targeting sites to perform site-specific recombination mediated cassette exchange. The corresponding vectors, host cells, and transgenic animals are claimed.

L27 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:391889 CAPLUS <<LOGINID::20070807>>
DN 136:396975
TI Construction of recombinant vectors comprising a transcriptionally silent element for conditional gene inactivation in mammalian cells
IN Xin, Hong-Bo; Kotlikoff, Michael

PA Cornell Research Foundation, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040685	A2	20020523	WO 2001-US43916	20011116
WO 2002040685	A3	20030109		
WO 2002040685	A9	20030724		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002019841	A5	20020527	AU 2002-19841	20011116
US 2004077089	A1	20040422	US 2003-416995	20031106
PRAI US 2000-249200P	P	20001116		
WO 2001-US43916	W	20011116		

AB The present invention provides recombinant vectors and methods of using the vectors in a high-throughput genetic system to rapidly generate conditional and/or conventional knockout mutants, such as mouse, useful to identify and define mammalian gene function in vivo. The methods of the invention combine ***gene*** ***trapping*** , ***gene*** targeting, and site-specific recombination techniques. The vectors comprise a transcriptionally silent genetic element that is inserted within a gene in a target cell in a manner that retains the functionality of the gene, and which element can be manipulated to inactivate the gene when desired. The vectors of the invention may be introduced ot cells via any means including non-biol. means, e.g., electroporation, or biol. means, e.g., via infection with a viral vector such as a retroviral vector.

L27 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable ***gene*** ***trap*** using the Cre/mutated ***lox*** system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (July, 1999) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB The ***gene*** ***trap*** technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of ***gene*** ***trapping*** is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the ***gene*** ***trap*** strategy. Here, we have constructed a new ***gene*** ***trap*** vector, pU-Hachi, employing the Cre-mutated ***lox*** system (Araki et al., 1997), in which a pair of mutant ***lox*** , lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (SA)-lox71-internal ribosomal entry site (***IRES***)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the trap vector. By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the trap vector is lost through integration into the genome. Thus, this method, termed exchangeable ***gene*** ***trapping*** , has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

=> d his

(FILE 'HOME' ENTERED AT 19:09:44 ON 07 AUG 2007)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:09:57 ON 07 AUG 2007
L1 696 S TRAP? (3A) VECTOR
L2 7 S L1 AND IRES AND LOX
L3 6 DUP REM L2 (1 DUPLICATE REMOVED)
L4 59 S L1 AND SPLICE ACCEPTOR
L5 4 S L4 AND LOX
L6 4 DUP REM L5 (0 DUPLICATES REMOVED)
L7 14 S L1 AND LOX
L8 11 DUP REM L7 (3 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 19:17:21 ON 07 AUG 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:18:33 ON 07 AUG 2007
L9 4930 S TRAP? AND VECTOR
L10 20 S L9 AND LOX
L11 15 DUP REM L10 (5 DUPLICATES REMOVED)
L12 21182 S INSERTION? (3A)(MUTATION OR MUTAGEN?)
L13 30 S L12 AND LOX
L14 23 DUP REM L13 (7 DUPLICATES REMOVED)
L15 3 S SPLICE ACCEPTOR AND IRES AND LOX
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)
L17 2872 S SPLICE ACCEPTOR
L18 25991 S L12 OR L9
L19 146 S L18 AND L17
L20 19 S L19 AND IRES
L21 19 DUP REM L20 (0 DUPLICATES REMOVED)
L22 18 S IRES AND LOX
L23 15 DUP REM L22 (3 DUPLICATES REMOVED)
L24 2751 S GENE (3A) TRAP?
L25 33 S L24 AND LOX
L26 8 S L25 AND IRES
L27 7 DUP REM L26 (1 DUPLICATE REMOVED)

=> dup rem l33
L33 IS NOT VALID HERE
The L-number entered has not been defined in this session, or it has been deleted. To see the L-numbers currently defined in this session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem l25
PROCESSING COMPLETED FOR L25
L28 26 DUP REM L25 (7 DUPLICATES REMOVED)

=> s l28 not l27
L29 20 L28 NOT L27

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 20 ANSWERS - CONTINUE? Y/(N):y

L29 ANSWER 1 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:248868 BIOSIS <<LOGINID::20070807>>
DN PREV200510036473
TI Characterization of an exchangeable ***gene*** ***trap*** using pU-17 carrying a stop codon-beta geo cassette.
AU Taniwaki, Takuya; Haruna, Kyoko; Nakamura, Hiroshi; Sekimoto, Tomohisa; Oike, Yuichi; Imaizumi, Takashi; Saito, Fumiyo; Muta, Mayumi; Soejima, Yumi; Utoh, Ayako; Nakagata, Naomi; Araki, Masatake; Yamamura, Ken-ichi [Reprint Author]; Kimi, Araki
CS Kumamoto Univ, Inst Mol Embryol and Genet, Kuhonji 4-24-1, Kumamoto 8620976, Japan
yamamura@gpo.kumamoto-u.ac.jp; arakimi@gpo.kumamoto-u.ac.jp
SO Development Growth & Differentiation, (APR 2005) Vol. 47, No. 3, pp. 163-172.
CODEN: DGDFA5. ISSN: 0012-1592.
DT Article
LA English
ED Entered STN: 8 Jul 2005
Last Updated on STN: 8 Jul 2005
AB We have developed a new exchangeable ***gene*** ***trap*** vector, pU-17, carrying the intron-lox71-splicing acceptor (SA)-beta geo-loxP-pA-lox2272-pSP73-lox511. The SA contains three stop codons in-frame with the ATG of beta galactosidase/neomycin-resistance fusion gene (beta geo) that can function in promoter trapping. We found that the trap vector was highly selective for integrations in the introns adjacent to the exon containing the start codon. Furthermore, by using the Cre-mutant ***lox*** system, we successfully replaced the beta geo gene with the enhanced green fluorescent protein (EGFP) gene, established mouse lines with the replaced clones, removed the selection marker gene by mating with Flp-deleter mice, and confirmed that the replaced EGFP gene was expressed in the same pattern as the beta geo gene. Thus, using this pU-17 trap vector, we can initially carry out random mutagenesis, and then convert it to a gain-of-function mutation by replacing the beta geo gene with any gene of interest to be expressed under the control of the trapped promoter through Cre-mediated recombination.

L29 ANSWER 2 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:240214 BIOSIS <<LOGINID::20070807>>
DN PREV200300240214
TI Generation and genetic modification of dendritic cells derived from mouse embryonic stem cells.
AU Senju, Satoru; Hirata, Shinya; Matsuyoshi, Hidetake; Masuda, Masako; Uemura, Yasushi; Araki, Kimi; Yamamura, Ken-ichi; Nishimura, Yasuharu [Reprint Author]
CS Division of Immunogenetics, Department of Neuroscience and Immunology, Graduate School of Medical Sciences, Kumamoto University, 2-2-1 Honjo, Kumamoto, 860-0811, Japan
mxnishim@gpo.kumamoto-u.ac.jp

SO Blood, (May1 2003) Vol. 101, No. 9, pp. 3501-3508. print.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 21 May 2003
Last Updated on STN: 21 May 2003
AB We developed a method to generate dendritic cells (DCs) from mouse embryonic stem (ES) cells. We cultured ES cells for 10 days on feeder cell layers of OP9, in the presence of granulocyte-macrophage colony-stimulating factor in the latter 5 days. The resultant ES cell-derived cells were transferred to bacteriologic Petri dishes without feeder cells and further cultured. In about 7 days, irregularly shaped floating cells with protrusions appeared and these expressed major histocompatibility complex class II, CD11c, CD80, and CD86, with the capacity to stimulate primary mixed lymphocyte reaction (MLR) and to process and present protein antigen to T cells. We designated them ES-DCs (ES cell-derived dendritic cells), and the functions of ES-DCs were comparable with those of DCs generated from bone marrow cells. Upon transfer to new dishes and stimulation with interleukin-4 plus tumor necrosis factor alpha, combined with anti-CD40 monoclonal antibody or lipopolysaccharide, ES-DCs completely became mature DCs, characterized by a typical morphology and higher capacity to stimulate MLR. Using an expression vector containing the internal ribosomal entry site-puromycin N-acetyltransferase gene or a Cre- ***lox*** -mediated exchangeable ***gene*** - ***trap*** system, we could efficiently generate ES cell transfectants expressing the products of introduced genes after their differentiation to DCs. ES-DCs expressing invariant chain fused to a pigeon cytochrome C epitope presented the epitope efficiently in the context of Ek. We primed ovalbumin (OVA)-specific cytotoxic T lymphocytes in vivo by injecting mice with ES-DCs expressing OVA, thus demonstrating immunization with ES-DCs genetically engineered to express antigenic protein. The methods may be applicable to immunomodulation therapy and ***gene*** - ***trap*** investigations of DCs.

L29 ANSWER 3 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:423540 BIOSIS <<LOGINID::20070807>>
DN PREV200200423540
TI Novel approaches for identifying genes regulating lymphocyte development and function.
AU Loy, Adele L. [Reprint author]; Goodnow, Christopher C. [Reprint author]
CS Genetics Laboratory and Medical Genome Centre, John Curtin School of Medical Research, Australian Cancer Research Foundation (ACRF), Australian National University, Canberra, ACT, 2601, Australia
adele.loy@anu.edu.au; chris.goodnow@anu.edu.au
SO Current Opinion in Immunology, (April, 2002) Vol. 14, No. 2, pp. 260-265. print.
CODEN: COPIEL. ISSN: 0952-7915.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 7 Aug 2002
Last Updated on STN: 7 Aug 2002

L29 ANSWER 4 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:430677 BIOSIS <<LOGINID::20070807>>
DN PREV200100430677
TI Exchangeable ***gene*** ***trap*** using a "double ***lox*** " targeting strategy mediated by Cre.
AU Araki, Kimi [Reprint author]; Kimura, Yumi; Muta, Mayumi; Saito, Fumiyo; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Dept. Dev. Genet., Kumamoto Univ., Kumamoto, Japan
SO Development Growth and Differentiation, (July, 2001) Vol. 43, No. Supplement, pp. S61. print.
Meeting Info.: 14th International Congress of Developmental biology. Kyoto, Japan. July 08-12, 2001. International Society of Developmental Biology; Japanese Society of Developmental Biologists.
CODEN: DGDFA5. ISSN: 0012-1592.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

L29 ANSWER 5 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:427935 BIOSIS <<LOGINID::20070807>>
DN PREV200100427935
TI Production and analyses of mutant lines established by exchangeable ***gene*** ***trap*** strategy.
AU Taniwaki, Takuya [Reprint author]; Sekimoto, Akihisa [Reprint author]; Saitou, Fumiyo; Imaizumi, Takashi; Sakumura, Yumi; Kimura, Yumi; Muta, Mayumi; Nakasima, Tatsuyuki; Utou, Ayako; Niizato, Haruko; Araki, Kimi [Reprint author]; Yamamura, Ken-ichi [Reprint author]; Araki, Masatake
CS Division of Developmental Genetics, IMEG, Kumamoto University School of Medicine, Kumamoto, Japan
SO Development Growth and Differentiation, (July, 2001) Vol. 43, No. Supplement, pp. S61. print.
Meeting Info.: 14th International Congress of Developmental biology. Kyoto, Japan. July 08-12, 2001. International Society of Developmental Biology; Japanese Society of Developmental Biologists.
CODEN: DGDFA5. ISSN: 0012-1592.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

L29 ANSWER 6 OF 20 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1997:53739 BIOSIS <<LOGINID::20070807>>
DN PREV199799352942
TI Recombinase-mediated-cassette-exchange (RMCE): A novel technique for integration of single copy of transgenes at pre-determined chromosomal sites. Application to the study of the human beta-globin LCR.
AU Bouhassira, E. E. [Reprint author]; Westerman, K.; Rochlin, I.; Nagel, R. L.; Leboulch, P.
CS A. Einstein Coll. Med., Bronx, NY, USA
SO Blood, (1996) Vol. 88, No. 10 SUPPL. 1 PART 1-2, pp. 190A.
Meeting Info.: Thirty-eighth Annual Meeting of the American Society of Hematology. Orlando, Florida, USA. December 6-10, 1996.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 4 Feb 1997
Last Updated on STN: 4 Feb 1997

L29 ANSWER 7 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:405865 CAPLUS <<LOGINID::20070807>>
DN 144:481709
TI Exchangeable ***gene*** ***trapping***
AU Araki, Kimi
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kumamoto, Japan
SO Genetically Engineered Mice Handbook (2006), 131-142. Editor(s): Sundberg, John P.; Ichiki, Tsutomu. Publisher: CRC Press LLC, Boca Raton, Fla.
CODEN: 69IBMO; ISBN: 978-0-8493-2220-4
DT Conference; General Review
LA English
AB A review discusses the principle, advantages, and disadvantages of ***gene*** ***trapping*** , with emphasis on the Cre-mutant ***Lox*** site-specific integration system and the pU-17 exchangeable trap vector.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 8 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:1232126 CAPLUS <<LOGINID::20070807>>
DN 144:461336
TI Cancer biology and transgenic technology in the mouse: Bridging the functional gap
AU McKinney, Cindy E.; Shashikant, Cooduvalli S.
CS Department of Animal Sciences, The Pennsylvania State University, University Park, PA, USA
SO Oncogenomics Handbook (2005), 303-321. Editor(s): LaRochelle, William J.; Shimkets, Richard A. Publisher: Humana Press Inc., Totowa, N. J.
CODEN: 69HORT; ISBN: 1-58829-425-0
DT Conference; General Review
LA English
AB A review. Cancer research has benefited from the ability to manipulate the mouse genome. Transgenic technol. is being used to produce mice with refined genomic mutations that recapitulate cancer development in the human. Transgenic mice have proven valuable in vivo models to address the biol. consequences of mutations found in tumors and interactions of two mutations in the same mouse. Carcinogen screening or therapeutic assessment might be enhanced by conducting 2-yr bioassays in the background of a mutant mouse model. The introduction of transgenic methods to limit mutant gene expression to a single tissue or cell type by Tet-inducible or Cre recombinase- ***Lox*** P technol. permits genetic models of otherwise lethal phenotypes to be developed. ***Gene*** ***traps*** in embryonic stem (ES) cells are being used to capture genes and conduct initial screens for gene function in "knockout" mice. Gene "knockdowns" in mouse embryos by lentivirus delivery of small interfering RNA (siRNA) constructs are poised to become valuable tools for studying cancer gene function with some addnl. technol. development. Transgenic mice allow a combinatorial approach utilizing genomic information and phys. manipulation of selected genes to dissect pathways altered during tumorigenesis. Finally, although transgenic mouse models are invaluable tools for dissecting cancer and related processes, future goals are to develop "humanized" transgenic mouse models that may more accurately respond to and reflect the human condition.
RE.CNT 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 9 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:571003 CAPLUS <<LOGINID::20070807>>
DN 143:72779
TI Recombinase mediated ***gene*** ***traps*** in plant cells
IN Gordon-Kamm, William J.; Lyznik, L. Alexander; Scelonge, Christopher J.; Klein, Theodore M.

PA Pioneer Hi-Bred International, Inc., USA; E. I. Dupont De Nemours and Company
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005059148	A1	20050630	WO 2004-US42232	20041216
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005144665	A1	20050630	US 2004-15612	20041217

PRAI US 2003-530402P P 20031217
AB The present invention provides methods for identifying a genetically modified plant cell by using recombinase-mediated ***gene*** ***traps*** . Recombinase-mediated cassette exchange resulted in activation of two marker genes in the donor locus or target locus by using two independent recombination systems. FLP or Cre recombinase and FRT or ***Lox*** sites are used in the recombinase mediated ***gene*** ***traps*** .
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 10 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:109968 CAPLUS <<LOGINID::20070807>>
DN 142:349687
TI ***Gene*** ***trap*** and ***gene*** inversion methods for conditional gene inactivation in the mouse
AU Xin, Hong-Bo; Deng, Ke-Yu; Shui, Bo; Qu, Shimian; Sun, Qi; Lee, Jane; Greene, Kai Su; Wilson, Jason; Yu, Ying; Feldman, Morris; Kotlikoff, Michael I.
CS Department of Biomedical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY, 14853, USA
SO Nucleic Acids Research (2005), 33(2), e14/1-e14/10
CODEN: NARHAD; ISSN: 0305-1048
PB Oxford University Press
DT Journal
LA English
AB Conditional inactivation of individual genes in mice using site-specific recombinases is an extremely powerful method for detg. the complex roles of mammalian genes in developmental and tissue-specific contexts, a major goal of post-genomic research. However, the process of generating mice with recombinase recognition sequences placed at specific locations within a gene, while maintaining a functional allele, is time consuming, expensive and tech. challenging. We describe a system that combines ***gene*** ***trap*** and site-specific DNA inversion to generate mouse embryonic stem (ES) cell clones for the rapid prodn. of conditional knockout mice, and the use of this system in an initial ***gene*** ***trap*** screen. ***Gene*** . ***trapping*** should allow the selection of thousands of ES cell clones with defined insertions that can be used to generate conditional knockout mice, thereby providing extensive parallelism that eliminates the time-consuming steps of targeting vector construction and homologous recombination for each gene.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 11 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:9198 CAPLUS <<LOGINID::20070807>>
DN 142:91478
TI Gene expression profiles in rheumatoid arthritis and osteoarthritis and their use in diagnosis and monitoring disease progress
IN Blaess, Stefan
PA Germany
SO Ger. Offen., 89 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 10328033	A1	20050105	DE 2003-10328033	20030619

PRAI DE 2003-10328033 20030619
AB DNA microarrays that can be used to diagnose and monitor rheumatoid arthritis (RA) and osteoarthritis (OA) are described. Gene expression is analyzed using software that can compare m-dimensional gene expression profiles multi-parametrical with n-dimensional ref. gene expression profiles for diagnostics, sub diagnostics classification and therapy decisions.

L29 ANSWER 12 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:756907 CAPLUS <<LOGINID::20070807>>
DN 141:275706
TI DNA microarray analysis of gene expression in the diagnosis of estrogen receptor positive- and negative-breast cancer
IN Erlander, Mark G.; Ma, Xiao-Jun; Wang, Wei; Wittliff, James L.
PA Arcturus Bioscience, Inc., USA
SO PCT Int. Appl., 226 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004079014	A2	20040916	WO 2004-US6736	20040304
WO 2004079014	A3	20050331		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2004079014	A2	20040916	WO 2002-XA2004006736	20040304
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005208500	A1	20050922	US 2004-794263	20040304
EP 1599607	A2	20051130	EP 2004-717480	20040304
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
JP 2006519620	T	20060831	JP 2006-509162	20040304

PRAI US 2003-451942P P 20030304
WO 2004-US6736 A 20040304
AB The invention relates to the identification and use of gene expression profiles, or patterns, suitable for identification of populations that are pos. and neg. for estrogen receptor expression. The gene expression profiles may be embodied in nucleic acid expression, protein expression, or other expression formats, and may be used in the study and/or diagnosis of cells and tissue in breast cancer as well as for the study and/or detn. of prognosis of a patient, including breast cancer survival.

L29 ANSWER 13 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:515644 CAPLUS <<LOGINID::20070807>>
DN 141:65052
TI Methods for the identification, assessment, and treatment of patients with proteasome inhibition therapy
IN Mulligan, George; Bryant, Barbara M.; Morrissey, Michael P.; Bolt, Andrew; Damokosh, Andrew I.
PA Millennium Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 178 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004053066	A2	20040624	WO 2003-US38539	20031204
WO 2004053066	A3	20060908		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2508348	A1	20040624	CA 2003-2508348	20031204
AU 2003298873	A1	20040630	AU 2003-298873	20031204
US 2004156854	A1	20040812	US 2003-728055	20031204
EP 1581629	A2	20051005	EP 2003-796633	20031204
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006517093	T	20060720	JP 2004-559278	20031204
MX 2005PA05923	A	20050921	MX 2005-PA5923	20050602

PRAI US 2002-431514P P 20021206
WO 2003-US38539 W 20031204
AB The present invention is directed to the identification of markers that can be used to det. whether patients with cancer are clin. responsive or non-responsive to a therapeutic regimen prior to treatment. In particular, the present invention is directed to the use of certain combinations of markers, wherein the expression of the markers correlates with responsiveness or non-responsiveness to a therapeutic regimen comprising proteasome inhibition. Thus, by examg. the expression levels

of individual markers and those comprising a marker set, it is possible to det. whether a therapeutic agent, or combination of agents, will be most likely to reduce the growth rate of tumors in a clin. setting.

L29 ANSWER 14 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:133066 CAPLUS <<LOGINID::20070807>>
DN 141:18366
TI FISH analysis comparing genome organization in the domestic horse (Equus caballus) to that of the Mongolian wild horse (E. przewalskii)
AU Myka, J. L.; Lear, T. L.; Houck, M. L.; Ryder, O. A.; Bailey, E.
CS M.H. Gluck Equine Research Center, Department of Veterinary Science, University of Kentucky, Lexington, KY, 40546-0099, USA
SO Cytogenetic and Genome Research (2003), 102(1-4), 222-225
CODEN: CGRYAJ; ISSN: 1424-8581
PB S. Karger AG
DT Journal
LA English
AB Przewalski's wild horse (E. przewalskii, EPR) has a diploid chromosome no. of 2n = 66 while the domestic horse (E. caballus, ECA) has a diploid chromosome no. of 2n = 64. Discussions about their phylogenetic relationship and taxonomic classification have hinged on comparisons of their skeletal morphol., protein and mitochondrial DNA similarities, their ability to produce fertile hybrid offspring, and on comparison of their chromosome morphol. and banding patterns. Previous studies of GTG-banded karyotypes suggested that the chromosomes of both equids were homologous and the difference in chromosome no. was due to a Robertsonian event involving two pairs of acrocentric chromosomes in EPR and one pair of metacentric chromosomes in ECA (ECA5). To det. which EPR chromosomes were homologous to ECA5 and to confirm the predicted chromosome homologies based on GTG banding, the authors constructed a comparative gene map between ECA and EPR by FISH mapping 46 domestic horse-derived BAC clones contg. genes previously mapped to ECA chromosomes. The results indicated that all ECA and EPR chromosomes were homologous as predicted by GTG banding, but provide new information in that the EPR acrocentric chromosomes EPR23 and EPR24 were shown to be homologues of the ECA metacentric chromosome ECA5.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:238349 CAPLUS <<LOGINID::20070807>>
DN 138:216479
TI method for selecting a clone of a cell library containing mutation introduced by ***gene*** ***trapping***
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO Eur. Pat. Appl., 17 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1295951	A1	20030326	EP 2001-308108	20010924
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
PRAI EP 2001-308108		20010924		

AB This invention provides a method for selecting a clone of a cell library contg. a mutation in a gene that is expressed in a test cell. The method comprising: providing cDNA obtained by reverse transcription of mRNA of the test cell; providing a collection of cultured cells of said library organized into individual clones, wherein each clone is of a cell having a mutation in an exon in its genome, the mutation being in a different exon in cells of different clones; providing an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations in; exposing the cDNA to the array under conditions permitting hybridization of polynucleotides in the array to nucleic acids; detecting hybridization of cDNA to a polynucleotide on the array; and selecting a clone in the collection from which a hybridizing polynucleotide detected at is an exon fragment. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon trap vector for mutating ES cells.
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:961831 CAPLUS <<LOGINID::20070807>>
DN 138:249000
TI Large scale ***gene*** ***trap*** mutagenesis
AU Araki, Masatake; Araki, Kimi
CS Institute of Genetic Experiment, Kumamoto University, Japan
SO Igaku no Ayumi (2002), 203(3), 201-205
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku Shuppan
DT Journal; General Review
LA Japanese
AB A review. The ***gene*** ***trap*** method for mutagenesis of embryonic stem cells by using promoter-less trap vectors was discussed. The outlines of the strategies of gene targeting were described by covering the topics on vector design and establishing transgenic mice lines. The genetic substitution by the ***trap*** technique and prodn. ***gene*** knockin mice and control of the transgene expression by the Cre/ ***lox*** system were also described. The impact of application of the trap method to the large scale prodn. of transgenic animal models for human diseases was discussed.

L29 ANSWER 17 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:783626 CAPLUS <<LOGINID::20070807>>
DN 138:67446
TI Site-directed integration of the cre gene mediated by Cre recombinase using a combination of mutant ***lox*** sites
AU Araki, Kimi; Araki, Masatake; Yamamura, Ken-ichi
CS Institute of Molecular Embryology and Genetics, Department of Developmental Genetics, Kumamoto University, Kumamoto, 862-0976, Japan
SO Nucleic Acids Research (2002), 30(19), e103/1-e103/8
CODEN: NARHAD; ISSN: 0305-1048
PB Oxford University Press
DT Journal
LA English
AB The Cre- ***lox*** system is an important tool for genetic manipulation. To promote integrative reactions, two strategies using mutant ***lox*** sites have been developed. One is the left element/right element (LE/RE)-mutant strategy and the other is the cassette exchange strategy using heterospecific ***lox*** sites such as lox511 or lox2272. We compared the recombination efficiencies using these mutant ***lox*** sites in embryonic stem (ES) cells, and found that the combination of the LE/RE mutant and lox2272 showed high recombination efficiency and stability of the recombined structure. Taking advantage of this stability, we successfully integrated the cre gene into the mutant ***lox*** sites by Cre-mediated recombination. Germ line chimeric mice were produced from the cre-integrated ES cell clones, and Cre-expressing mouse lines were established. The inserted cre gene was stably maintained through the generations. This cre knock-in system using the LE/RE-lox2272 combination should be useful for the prodn. of various cre mice for ***gene*** targeting or ***gene*** ***trapping*** .
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 18 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:924281 CAPLUS <<LOGINID::20070807>>
DN 136:32674
TI Embryonic stem cell libraries indexed to nucleic acid microarrays
IN Ong, Christopher J.
PA The University of British Columbia, Can.
SO U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2001053524	A1	20011220	US 2001-883745	20010618
US 6867035	B2	20050315		
CA 2309371	A1	20011216	CA 2000-2309371	20000616
CA 2350976	A1	20011216	CA 2001-2350976	20010618
PRAI CA 2000-2309371	A	20000616		

AB The invention relates to libraries of embryonic stem cells in which the genome of members of the library are modified by ***gene*** ***trapping*** . This invention provides a method for selecting a clone of an ES cell contg. a mutation in a gene that is expressed in a test cell. The method comprising, obtaining cDNA of the test cell, providing a collection of cultured ES cells organized into individual clones, wherein each clone of an ES cell having a mutation in an exon in its genome. The invention further provides an array of different single stranded polynucleotides, the polynucleotides being fragments of exons contg. mutations; exposing the cDNA to the array under conditions permitting hybridization of polynucleotide. The invention relates to detection of hybridization of cDNA to a polynucleotide on the array and selecting a clone in the collection. This invention also provides a system for testing expression of a gene in a test cell. Also provided is a preferred exon trap vector for mutating ES cells.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:390639 CAPLUS <<LOGINID::20070807>>
DN 125:50249
TI Trapping of mammalian promoters by cre- ***lox*** site-specific recombination
AU Fukushige, Shinichi; Ikeda, Joh-E.
CS Ikeda GenoSPHERE Project, ERATO, Japan
SO DNA Research (1996), 3(2), 73-80
CODEN: DARSE8; ISSN: 1340-2838
PB Kazusa DNA Research Institute
DT Journal
LA English
AB One of the challenges in human genome research is to identify the promoter sequences which play a key role in the regulation of gene expression. We report here a new promoter trapping system for use with mammalian cells

comprised of the following three steps: (1) Cloning of DNA fragments into a promoter-trapping vector, (2) integration of the trapping vector into a designated target in the mammalian genome using the Cre-site-specific recombinase, and (3) screening of integrants for trapped promoter sequences by activation of the luciferase gene. To assess the efficiency of this system, ***lox*** trapping vectors contg. sense tk promoter, antisense tk promoter, or a non-promoter sequence of the neo gene were employed. The resulting levels of luciferase activity of the site specific integrants were measured directly. Luciferase activity of the integrants can be assayed under conventional culture conditions by simply replacing the culture medium with potassium phosphate buffer contg. lucifenin. Only those G418-resistant colonies carrying the tk promoter in the normal orientation exhibited a 21-35-fold increase in luciferase activity over that of the other integrants. These results indicate that this system is an effective means of trapping promoter sequences from random mammalian genomic DNA fragments.

L29 ANSWER 20 OF 20 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2005445137 EMBASE <<LOGINID::20070807>>
TI Functional genomics in the mouse.
AU Perkins A.S.
CS A.S. Perkins, Department of Pathology and Molecular, Cellular, Developmental Biology, Yale University, P.O. Box 208023, New Haven, CT 06520-8023, United States. archibald.perkins@yale.edu
SO Functional and Integrative Genomics, (2002) Vol. 2, No. 3, pp. 81-91. .
Refs: 65
ISSN: 1438-793X CODEN: FIGUBY
CY Germany
DT Journal; General Review
FS 022 Human Genetics
LA English
SL English
ED Entered STN: 28 Nov 2005
Last Updated on STN: 28 Nov 2005
AB The mouse is the premier genetic model organism for the study of human disease and development. With the recent advances in sequencing of the human and mouse genomes, there is strong interest now in large-scale approaches to decipher the function of mouse genes using various mutagenesis technologies. This review discusses what tools are currently available for manipulating and mutagenizing the mouse genome, such as ethylnitrosourea and ***gene*** ***trap*** mutagenesis, engineered inversions and deletions using the cre- ***lox*** system, and proviral insertional mutagenesis in somatic cells, and how these are being used to uncover gene function. .COPYRGT. Springer-Verlag 2002.

=> FIL STNGUIDE
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 339.52 423.17

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ENTRY SESSION
FULL ESTIMATED COST 0.24 423.41

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL
ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -74.88

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=> d his

(FILE 'HOME' ENTERED AT 19:09:44 ON 07 AUG 2007)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:09:57 ON 07 AUG 2007
L1 696 S TRAP? (3A) VECTOR
L2 7 S L1 AND IRES AND LOX
L3 6 DUP REM L2 (1 DUPLICATE REMOVED)
L4 59 S L1 AND SPLICE ACCEPTOR
L5 4 S L4 AND LOX
L6 4 DUP REM L5 (0 DUPLICATES REMOVED)
L7 14 S L1 AND LOX
L8 11 DUP REM L7 (3 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 19:17:21 ON 07 AUG 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:18:33 ON 07 AUG 2007
L9 4930 S TRAP? AND VECTOR
L10 20 S L9 AND LOX
L11 15 DUP REM L10 (5 DUPLICATES REMOVED)
L12 21182 S INSERTION? (3A)(MUTATION OR MUTAGEN?)
L13 30 S L12 AND LOX
L14 23 DUP REM L13 (7 DUPLICATES REMOVED)
L15 3 S SPLICE ACCEPTOR AND IRES AND LOX
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)
L17 2872 S SPLICE ACCEPTOR
L18 25991 S L12 OR L9
L19 146 S L18 AND L17
L20 19 S L19 AND IRES
L21 19 DUP REM L20 (0 DUPLICATES REMOVED)
L22 18 S IRES AND LOX
L23 15 DUP REM L22 (3 DUPLICATES REMOVED)
L24 2751 S GENE (3A) TRAP?
L25 33 S L24 AND LOX
L26 8 S L25 AND IRES
L27 7 DUP REM L26 (1 DUPLICATE REMOVED)
L28 26 DUP REM L25 (7 DUPLICATES REMOVED)
L29 20 S L28 NOT L27

FILE 'STNGUIDE' ENTERED AT 19:34:49 ON 07 AUG 2007

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 19:37:23 ON 07 AUG 2007

=> s l1 and (splice acceptor or sa)
L30 71 L1 AND (SPLICE ACCEPTOR OR SA)

=> s l30 and py<=1999
L31 23 L30 AND PY<=1999

=> dup rem l31
PROCESSING COMPLETED FOR L31
L32 14 DUP REM L31 (9 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 14 ANSWERS - CONTINUE? Y/(N):y

L32 ANSWER 1 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:764183 CAPLUS <<LOGINID::20070807>>
DN 132:19627
TI A ***vector*** for gene ***trap*** , and a method for gene ***trapping*** by using the ***vector*** in Drosophila melanogaster
IN Lukacsovich, Tamas; Asztalos, Zoltan; Yamamoto, Daisuke; Awano, Wakae
PA Japan Science and Technology Corp., Japan
SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9961604	A2	19991202	WO 1999-JP2683	19990521 <--
WO 9961604	A3	20000302		
W: CA, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
JP 11332564	A	19991207	JP 1998-141952	19980522 <--
JP 3688118	B2	20050824		
CA 2329141	A1	19991202	CA 1999-2329141	19990521 <--
CA 2329141	C	20070710		
EP 1078050	A2	20010228	EP 1999-921222	19990521
EP 1078050	B1	20061018		
R: DE, ES, FR, GB				
US 7026525	B1	20060411	US 2001-700843	20010202
PRAI JP 1998-141952	A	19980522		
WO 1999-JP2683	W	19990521		

AB The present application provides a ***vector*** for ***trapping*** an unknown gene of Drosophila melanogaster , which is a recombinant plasmid comprising the following nucleotide sequences in this order: an artificial consensus splicing acceptor site; a synthetic "stop/start" sequence; a reporter gene; a drug resistance gene; a gene responsible for a detectable phenotype of the Drosophila melanogaster ; and a synthetic splicing donor site. The present application also provides a method for trapping an unknown gene of Drosophila melanogaster by using the vector. This method involves introducing the vector into the genome of a white minus fly, selecting primary transformants resistant to a drug, crossing the primary transformants with a transposase source strain to force the vector to jump into other locations, selecting secondary transformants by picking up flies having strong eye color, crossing secondary transformants with UAS (Upstream Activator Sequence)-luciferase harboring strain and measuring the reporter gene expression of the resultant flies and identifying the trapped gene by cloning and sequencing the cDNAs fused to

the reporter gene and the gene responsible for a detectable phenotype of the fly. A heat-shock promoter is used. When the gene trap construct is being inserted into an intron of an endogenous gene, the marker genes of the construct are supposed to be spliced on mRNA level to the exons of the trapped gene by using artificial splicing acceptor and donor sites. Specifically, the Gal4 mRNA should be joint to the exon located upstream of the insertion site and at the same time the mini-white mRNA is fused to the following exon accomplishing the dual tagging of the trapped gen. This vector system has industrial applicability in that it offers an exceptional opportunity for easy and fast cloning of the gene responsible for the obsd. phenotype. Furthermore, by using the UAS-driven coding sequence of any gene of interest, that gene can be expressed in identical patterns than those of the trapped genes and these expressions can be regulated temporarily at any desired developmental stage.

L32 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:640997 CAPLUS <<LOGINID::20070807>>
DN 131:282372
TI Vectors containing 3' gene trap cassettes for gene mutagenesis and gene discovery
IN Zambrowicz, Brian; Friedrich, Glenn A.; Sands, Arthur T.
PA Lexicon Genetics Incorporated, USA
SO PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9950426	A1	19991007	WO 1999-US6474	19990326 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6080576	A	20000627	US 1998-57328	19980408
CA 2323834	A1	19991007	CA 1999-2323834	19990326 <--
AU 9932036	A	19991018	AU 1999-32036	19990326 <--
AU 751520	B2	20020815		
EP 1066392	A1	20010110	EP 1999-914126	19990326
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002509727	T	20020402	JP 2000-541314	19990326
JP 3725782	B2	20051214		
US 2005095713	A1	20050505	US 2004-916782	20040811
PRAI US 1998-79729P	P	19980327		
US 1998-57328	A	19980408		
US 1998-81727P	P	19980414		
US 1998-109302P	P	19981120		
US 1999-276533	A1	19990325		
WO 1999-US6474	W	19990326		
US 2002-158735	A1	20020529		

AB Novel vectors are described that incorporate, inter alia , a novel 3' gene trap cassette which can be used to efficiently trap and identify previously unknown cellular genes. Efficient methods of 3' gene trapping are provided that allow a greater percentage of genes in the target cell genome to be trapped and rapidly identified. The presently described 3' gene trap cassette comprises in operable combination, a promoter region, an exon (typically characterized by a translation initiation codon and open reading frame and/or internal ribosome entry site), a splice donor sequence, and, optionally, intronic sequences. The splice donor sequence is operatively positioned such that the exon of the 3' gene trap cassette is spliced to the ***splice*** ***acceptor*** site of a downstream exon or a cellularly encoded exon. In a preferred embodiment, the exon component of the 3' gene trap cassette, which also serves as a sequence acquisition cassette, will comprise exon sequence and a splice donor sequence derived from genetic material that naturally occurs in an eukaryotic cell. Addnl. embodiments of the present invention include recombinant vectors, particularly viral vectors, that have been genetically engineered to incorporate the 3' gene trap cassette. The vectors can also be engineered to include a 5' gene trap cassette that typically contains a ***splice*** ***acceptor*** site located 5' to an exon (which can encode a selectable marker gene) followed by an operatively positioned polyadenylation sequence. The splicing machinery is better able to recognize an exon type sequence present adjacent to or relatively close to a promoter when splicing into downstream exons. Vectors incorporating the described 3' gene trap cassette find particular application in gene discovery and in the prodn. of mutated cells and animals.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:64965 CAPLUS <<LOGINID::20070807>>
DN 130:120465
TI A two-component system for identification and tagging of genes showing tissue- or cell-limited expression
IN Ong, Christopher J.; Priatel, John J.; Jirik, Frank R.
PA The University of British Columbia, Can.
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9902719	A1	19990121	WO 1998-CA677	19980710 <--
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2205888	A1	19990111	CA 1997-2205888	19970711 <--
CA 2295475	A1	19990121	CA 1998-2295475	19980710 <--
EP 1003893	A1	20000531	EP 1998-933402	19980710
R: AT, BE, CH, DE, DK, FR, GB, IT, LI, NL, SE				
US 6777235	B1	20040817	US 1999-295464	19990419
PRAI CA 1997-2205888	A	19970711		
WO 1998-CA677	W	19980710		

AB Reporter genes and methods for their use that allow the detection and cloning of eucaryotic genes whose expression is restricted to certain tissues or specialized cell types are described. The method uses two reporter genes, one is under control of a promoter selected for its restricted function in a particular cell or tissue. The second gene is a promoter-less reporter gene that may have a 5'-terminal IRES element to allow in-frame translation of the transcript. In addn., there may be a ***splice*** ***acceptor*** site upstream of the second gene to ensure that it is not lost during post-transcriptional processing. The two genes are introduced into target ells using independent vectors or transformations. Transformed cells are allowed to differentiate to produce a specialized cell or tissue that is monitored for expression of both reporter genes, thereby detecting a gene into which the ***trap*** ***vector*** has integrated which is expressed in the same cell or tissue type as the selected promoter. Preferably, the two reporter gene products are subunits of a single protein that do not have sep. functions, e.g. the .alpha.-complementing and .omega. peptides of .beta.-galactosidase. Alternatively, two-hybrid systems can be used with expression of both components necessary or expression of a reporter gene. A system depending upon the interaction of aequorin and green fluorescent protein is also described.
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:439294 CAPLUS <<LOGINID::20070807>>
DN 131:69280
TI Novel gene trap and its use for high efficiency selection of regulated eukaryotic genes
IN Baetscher, Manfred; Nir, Waan-jeng
PA Biotransplant, Inc., USA
SO U.S., 23 pp., Cont. of U.S. Ser. No. 374,833, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5922601	A	19990713	US 1996-716854	19960916 <--
PRAI US 1995-374833	B1	19950119		

AB The invention provides a novel gene trap construct that allows for high efficiency identification and selection of eukaryotic genes whose activity is regulated upon a cellular transition. Said viral vector comprises in its downstream sequence (i) a cassette having a functional ***splice*** ***acceptor*** , a translation stop sequence and an internal ribosome entry site and (ii) a promoterless protein coding sequence encoding at least one polypeptide providing pos. and neg. selection traits. Also provided is a method for identification of genes whose activity is regulated upon a cellular transition event by introducing the gene trap construct into a cell and observing expression of the pos. and/or neg. selection traits before and after the transition event.
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 5 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2000:433494 BIOSIS <<LOGINID::20070807>>
DN PREV200000433494
TI Exchangeable gene trap using the Cre/mutated lox system.
AU Araki, Kimi; Imaizumi, Takashi; Sekimoto, Tomohisa; Yoshinobu, Kumiko; Yoshimuta, Junichiro; Akizuki, Miwa; Miura, Katsutaka; Araki, Masatake; Yamamura, Ken-ichi [Reprint author]
CS Institute of Molecular Embryology and Genetics, Kumamoto University School of Medicine, Kuhonji 4-24-1, Kumamoto, 862-0976, Japan
SO Cellular and Molecular Biology (Noisy-Le-Grand), (***July, 1999***) Vol. 45, No. 5, pp. 737-750. print.
DT Article
LA English
ED Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002
AB The gene trap technique is a powerful approach for characterizing and mutating genes involved in mouse development. However, one shortcoming of

gene trapping is the relative inability to induce subtle mutations. This problem can be overcome by introducing a knock-in system into the gene trap strategy. Here, we have constructed a new gene ***trap*** ***vector*** , pU-Hachi, employing the Cre-mutated lox system (Araki et al., 1997), in which a pair of mutant lox, lox71 and lox66, was used to promote targeted integrative reaction by Cre recombinase. The pU-Hachi carries splicing acceptor (***SA***)-lox71-internal ribosomal entry site (IRES)-beta-geo-pA-loxP-pA-pUC. By using this vector, we can carry out random insertional mutagenesis as the first step, and then we can replace the beta-geo gene with any gene of interest through Cre-mediated integration. We have isolated 109 trap clones electroporated with pU-Hachi, and analyzed their integration patterns by Southern blotting to select those carrying a singlecopy of the ***trap*** ***vector*** . By use of some of these clones, we have succeeded in exchanging the reporter gene at high efficiency, ranging between 20-80%. This integration system is also quite useful for plasmid rescue to recover flanking genomic sequences, because a plasmid vector sequence can be introduced even when the pUC sequence of the ***trap*** ***vector*** is lost through integration into the genome. Thus, this method, termed exchangeable gene trapping, has many advantages as the trapped clones can be utilized to express genes with any type of mutation.

L32 ANSWER 6 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
AN 1999:464732 BIOSIS <<LOGINID::20070807>>
DN PREV199900464732
TI Def-2, -3, -6 and -8, novel mouse genes differentially expressed in the haemopoietic system.
AU Hotfilder, Marc; Baxendale, Sarah; Cross, Michael A.; Sablitzky, Fred (Reprint author)
CS Department of Medicine, Windeyer Institute of Medical Sciences, University College London, 46 Cleveland Street, London, W1P 6DB, UK
SO British Journal of Haematology, (***Aug., 1999***) Vol. 106, No. 2, pp. 335-344. print.
CODEN: BJHEAL. ISSN: 0007-1048.
DT Article
LA English
ED Entered STN: 1 Nov 1999
Last Updated on STN: 1 Nov 1999
AB To identify developmentally regulated genes during myeloid differentiation, a self-inactivating retroviral gene- ***trap*** ***vector*** carrying a beta-galactosidase-neomycin (***SA*** /lacZ/neo) fusion gene was constructed and used to infect myeloid progenitor cells (FDCP-Mix A4). G418-resistant and beta-galactosidase positive cell lines (gene-trap integration (GTI) clones) were established and induced to differentiate in vitro into either macrophages or granulocytes. Expression of the trapped loci was monitored at a single-cell level by analysing the mature cell types for beta-galactosidase activity. All 37 GTI clones tested showed down-regulation either during granulocyte or both granulocytic and macrophage differentiation. The endogenous coding regions fused to the ***SA*** /lacZ/neo reporter gene were isolated from eight clones. Molecular analysis revealed that half of them represented novel mouse genes (def-2, -3, -6 and -8) which we confirmed to be differentially expressed in primary haemopoietic tissues.Database searches revealed no significant similarities for def-2 (associated with haemopoietic progenitors) and def-8 (expressed most strongly in peripheral leucocytes). Def-6, which is down-regulated upon the differentiation into myeloid as well as erythroid lineages, was found to be closely related but not identical with the recently described B-cell-specific switch recombinase SWAP-70. Def-3, which is down-regulated upon differentiation into granulocytes but expressed in progenitor cells and macrophages, defines a novel family of RNA binding proteins.

L32 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:534939 CAPLUS <<LOGINID::20070807>>
DN 129:145633
TI Expression vectors for eukaryotic hosts for the trapping of genes for secreted proteins
IN Skarnes, William C.
PA University of Edinburgh, UK
SO U.S., 10 pp., Cont.-in-part of U.S. Ser. No. 404,727.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5789653	A	19980804	US 1995-497076	19950630 <--
US 5767336	A	19980616	US 1995-404727	19950315 <--
CA 2166850	A1	19960711	CA 1996-2166850	19960109 <--
CA 2166850	C	19990504		
AU 9640873	A	19960718	AU 1996-40873	19960109 <--
AU 673650	B2	19961114		
EP 731169	A1	19960911	EP 1996-300152	19960109 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 08308576	A	19961126	JP 1996-29792	19960110 <--
JP 3592426	B2	20041124		
PRAI GB 1995-423	A	19950110		
US 1995-404727	A2	19950315		
US 1995-497076	A	19950630		

AB Cloning and expression vectors that can be used to trap genes for secreted proteins are described. The vectors encode a type II transmembrane domain and a secretory lumen-sensitive indicator marker, i. e. one not easily detectable when the protein is sequestered in a structure such as the Golgi body, and optionally, a selectable marker and an exon- ***splice*** ***acceptor*** site. The gene isolation methods involve stably introducing the secretory trap vectors into an endogenous gene whereby the expression of the resultant fusion protein provides a differential expression of the indicator marker depending on whether the endogenous gene provides an N-terminal signal sequence. Use of the method with a lacZ reporter gene to clone genes for secreted proteins of embryonic stem cells is reported. Homologs of the sek and netrin-1 genes were obtained.
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:649803 CAPLUS <<LOGINID::20070807>>
DN 125:267525
TI Vectors and use thereof for capturing target genes encoding extracellular proteins
IN Skarnes, William C.
PA University of Edinburgh, UK
SO Can. Pat. Appl., 26 pp.
CODEN: CPXXEB
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI CA 2166850	A1	19960711	CA 1996-2166850	19960109 <--
CA 2166850	C	19990504		
US 5767336	A	19980616	US 1995-404727	19950315 <--
US 5789653	A	19980804	US 1995-497076	19950630 <--
PRAI GB 1995-423	A	19950110		
US 1995-404727	A	19950315		
US 1995-497076	A	19950630		

AB The invention relates to secretory gene trap vectors and methods of using such vectors to isolate extracellular proteins and to make cells and organisms with mutant secretory genes. The vectors encode a type II transmembrane domain and a lumen -sensitive indicator marker and optionally, a selectable marker and an exon- ***splice*** ***acceptor*** site. The gene isolation methods involve stably introducing the secretory trap vectors into an endogenous gene whereby the expression of the resultant fusion provides a differential expression of the indicator marker depending on whether the endogenous gene provides an N-terminal signal sequence. Plasmids encoding the CD4 transmembrane II domain fused to the .beta.geo chimeric gene were prepd. When inserted into a gene lacking a signal sequence, the resulting fusion protein behaves as a type II membrane protein, placing .beta.geo in the endoplasmic reticulum lumen where the .beta.gal enzyme in inactive. When inserted into a gene contg. a signal sequence, the resulting fusion protein acts like a type I membrane protein and the .beta.geo remains in the cytosol where it is active. Genes encoding a novel cadherin, an unc6-related laminin (netrin-1), the sek receptor tyrosine kinase, and 2 receptor-linked protein tyrosine phosphatases LAR and PTP.kappa. were identified in mice embryonic stem cells in this way. Embryos and transgenic mice derived from these cells could be analyzed for the expression pattern of these genes.

L32 ANSWER 9 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 3
AN 1996:461949 BIOSIS <<LOGINID::20070807>>
DN PREV199699184305
TI Gene tapping in differentiating cell lines: Regulation of the lysosomal protease cathepsin B in skeletal myoblast growth and fusion.
AU Gogos, Joseph A. [Reprint author]; Thompson, Rachel; Lowry, William; Sloane, Bonnie F.; Weintraub, Harold; Horwitz, Marshall
CS Cent. Neurobiol. Behavior, Coll. Phys. Surg., Columbia Univ., 701 W. 168th St., New York, NY 10032, USA
SO Journal of Cell Biology, (***1996***) Vol. 134, No. 4, pp. 837-847.
CODEN: JCLBA3. ISSN: 0021-9525.
DT Article
LA English
ED Entered STN: 11 Oct 1996
Last Updated on STN: 11 Oct 1996
AB To identify genes regulated during skeletal muscle differentiation, we have infected mouse C2C12 myoblasts with retroviral gene trap vectors, containing a promoterless marker gene with a 5' ***splice*** ***acceptor*** signal. Integration of the vector adjacent to an actively transcribed gene places the marker under the transcriptional control of the endogenous gene, while the adjacent vector sequences facilitate cloning. The ***vector*** insertionally mutates the ***trapped*** locus and may also form fusion proteins with the endogenous gene product. We have screened several hundred clones, each containing a ***trapping*** ***vector*** integrated into a different endogenous gene. In agreement with previous estimates based on hybridization kinetics, we find that a large proportion of all genes expressed in myoblasts are regulated during differentiation. Many of these genes undergo unique temporal patterns of activation or repression during cell growth and myotube formation, and some show specific patterns of subcellular localization. The first gene we have identified with this strategy is the lysosomal cysteine protease cathepsin B. Expression from

the trapped allele is upregulated during early myoblast fusion and downregulated in myotubes. A direct role for cathepsin B in myoblast growth and fusion is suggested by the observation that the trapped cells deficient in cathepsin B activity have an unusual morphology and reduced survival in low-serum media and undergo differentiation with impaired cellular fusion. The phenotype is reproduced by antisense cathepsin B expression in parental C2C12 myoblasts. The cellular phenotype is similar to that observed in cultured myoblasts from patients with I cell disease, in which there is diminished accumulation of lysosomal enzymes. This suggests that a specific deficiency of cathepsin B could contribute to the myopathic component of this illness.

L32 ANSWER 10 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 4
AN 1996:575256 BIOSIS <<LOGINID::20070807>>
DN PREV199799289937
TI Unexpected behavior of a gene ***trap*** ***vector*** comprising a fusion between the Sh ble and the lacZ genes.
AU Camus, A.; Kress, C.; Babinet, C.; Barra, J. [Reprint author]
CS Dep. d'Immunol., Unite de Biol. du Developpement, URA CNRS 1960, Inst. Pasteur, 25 rue du Dr Roux, 75724 Paris Cedex, France
SO Molecular Reproduction and Development, (***1996***) Vol. 45, No. 3, pp. 255-263.
CODEN: MREDEE. ISSN: 1040-452X.
DT Article
LA English
ED Entered STN: 23 Dec 1996
Last Updated on STN: 11 Feb 1997
AB A new gene ***trap*** ***vector*** has been designed, comprised of a fusion between the Sh ble gene, which confers resistance to the antibiotic phleomycin, and the lacZ gene (phleal fusion gene). A synthetic ***splice*** ***acceptor*** , made of the yeast branchpoint followed by a pyrimidine-rich sequence of 27 nucleotides, is included at the 5' extremity. The linearized gene ***trap*** ***vector*** was introduced into mouse embryonic stem cells (ES cells), and 40 phleomycin resistant (phle-r) cell lines possessing a single copy of the insert were selected. They were stable in expressing the lacZ gene. Reporter gene expression was studied at days 8.5 and 10.5 of embryonic development in chimeric embryos obtained after injection of phleo-r ES clones into 8-cell stage embryos. Out of 20 phleal lines examined, 14 exhibited beta-galactosidase expression at day 10.5. Use of the phleal fusion gene ***trap*** ***vector*** to select genes expressed in ES cells, therefore, is compatible with the isolation of genes expressed at midgestation. However, and most intriguingly, 10 out of these 14 cell lines (71%) displayed reporter gene expression mostly in heart and liver. Two of them exhibited, in addition, expression in central nervous system (CNS) or in CNS and limb buds, respectively. Germline chimeras were subsequently obtained and 15 mouse lines have been established. Intercrosses of animals heterozygous for the insertion revealed a mutant phenotype in several lines.

L32 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:911626 CAPLUS <<LOGINID::20070807>>
DN 124:1753
TI A lacZ-hygromycin fusion gene and its use in a gene ***trap*** ***vector*** for marking embryonic stem cells
AU Natarajan, Dipa; Boulter, Catherine A.
CS Dep. Genetics, Univ. Cambridge, Cambridge, CB2 3EH, UK
SO Nucleic Acids Research (***1995***), 23(19), 4003-4
CODEN: NARHAD; ISSN: 0305-1048
PB Oxford University Press
DT Journal
LA English
AB The authors constructed a gene ***trap*** ***vector*** , p.beta.gyg, which is based on pSA.beta.geo which carries a .beta.geo fusion gene downstream of the ***splice*** ***acceptor*** site from adenovirus major late transcript. Essentially, the neo gene and polyA site from pSA.beta.geo has been replaced with a hygromycin B phosphotransferase (hyg) gene and phosphoglycerate kinase polyA site from pKJ23 such that the hyg gene is in frame with the lacZ gene to generate the fusion gene .beta.gyg. The fusion gene is functional in mammalian cells, and the fusion protein retains biol. activity. This is the first report of hygromycin as a C-terminal fusion protein with .beta.-galactosidase.

L32 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:201900 CAPLUS <<LOGINID::20070807>>
DN 122:152668
TI Gene trap integrations in genes active in mouse embryonic stem cells efficiently detect developmentally regulated gene expression
AU Schuster-Gossler, Karin; Zachgo, Jochen; Soininen, Raija; Schoor, Michael; Korn, Reinhard; Gossler, Achim
CS Max-Delbruck-Laboratorium der MPG, Cologne, 50829, Germany
SO Transgenics (***1994***), 1(3), 281-91
CODEN: TADTEF; ISSN: 1023-6171
DT Journal
LA English
AB Two gene trap vectors were devised and used to detect patterns of gene expression during mouse postimplantation development. Constructs carry either a triple ***splice*** ***acceptor*** site and Kozak consensus sequence for efficient initiation of translation (pGTi) or a ***splice*** ***acceptor*** in front of a lacZ-neo fusion gene that lacks an ATG codon (pGTfu). With pGTfu in most cases significant proportions of endogenous genes were parts of fusion proteins, whereas with pGTi in most analyzed cases very little or no endogenous coding sequence was fused to lacZ. Vectors were introduced into mouse embryonic stem (ES) cells and chimeric embryos were produced by blastocyst injection. Reporter gene expression was monitored in days 8.5 and 12.5 chimeric embryos by staining for .beta.-galactosidase activity. Thirty-one cell lines that expressed the reporter gene as undifferentiated stem cells ("blue" lines) and 14 cell lines that did not show detectable staining in ES cells ("white" lines) were analyzed. Fifteen blue cell lines gave rise to staining in chimeras whereas the remaining 16 blue cell lines and all the white lines showed no .beta.-galactosidase activity in embryos at the analyzed stages. Four of the staining patterns were exclusively in extraembryonic tissues, 2 were widespread on days 8.5 and 12.5 and 9 cell lines (29% of the integrations active in ES cells) showed spatially and/or temporally regulated staining patterns. The results suggest that a significant proportion of genes expressed in ES cells show developmental regulation during postimplantation development and support the idea that genes with spatially and/or temporally regulated expression during postimplantation development can efficiently be identified by gene trap integrations in the genome of ES cells.

L32 ANSWER 13 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN DUPLICATE 5
AN 1992:368433 BIOSIS <<LOGINID::20070807>>
DN PREV199294050483; BA94:50483
TI A GENE TRAP APPROACH IN MOUSE EMBRYONIC STEM CELLS THE LACZ REPORTER IS
ACTIVATED BY SPLICING REFLECTS ENDOGENOUS GENE EXPRESSION AND IS MUTAGENIC
IN MICE.
AU SKARNES W C [Reprint author]; AUERBACH B A; JOYNER A L
CS DEP MOLECULAR MED GENETICS, UNIV TORONTO, TORONTO, CAN
SO Genes and Development, (***1992***) Vol. 6, No. 6, pp. 903-918.
CODEN: GEDEEP. ISSN: 0890-9369.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 9 Aug 1992
Last Updated on STN: 9 Aug 1992
AB We have confirmed that the gene ***trap*** ***vector*** pGT4.5 creates spliced fusion transcripts with endogenous genes and prevents the synthesis of normal transcripts at the site of integration. cDNA was prepared to the lacZ fusion transcript in three ES cell lines to recover endogenous exon sequences upstream of laZ. Each of the clones detected a unique-sized endogenous transcript, as well as the fusion transcript in the ES cell line from which the clone was derived. Sequence analysis of these clones and larger clones isolated from a random-primed cDNA library showed that the ***splice*** ***acceptor*** was used properly. For two insertions, the expression patterns of the lacZ reporter and the associated endogenous gene were compared in situ at three embryonic stages and were found to be similar. Three gene trap insertions were transmitted into the germ line, and abnormalities were observed with two of the three insertions in the homozygous state. RNA obtained from mice homozygous for the two mutant gene trap insertions was analyzed for nomal endogenous transcripts and negligible amounts were detected, indicating that littlr splicing around the gene trap insertion occurred. This work demonstrates the capacity of the gene ***trap*** ***vector*** to generate lacZ fusion transcripts, to accurately report endogenous gene expression, and to mutate the endogenous gene at the site of integration.

L32 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1991:37160 CAPLUS <<LOGINID::20070807>>
DN 114:37160
TI Exon trap cloning: using PCR to rapidly detect and clone exons from genomic DNA fragments
AU Auch, D.; Reth, M.
CS Max-Planck-Inst. Immunol., Freiburg, D-7800, Germany
SO Nucleic Acids Research (***1990***), 18(22), 6743-4
CODEN: NARHAD; ISSN: 0305-1048
DT Journal
LA English
AB A new method was developed which allows the selective and rapid cloning of exon sequences from large genomic DNA fragments independently of the expression level of their corresponding genes or availability of RNA. For this purpose the exon ***trap*** ***vector*** pL53In was constructed which was derived from pRSV-BC12-SEAP. PL53In contains the LTR of Rous Sarcoma Virus (RSV) as a strong promoter in front of a truncated 213 bp sequence of the phosphatase gene (the active site has been deleted) followed by the 3' part of the rat preproinsulin gene including 175 bp of the 2nd exon with a 5' splice donor site, 500 bp of intron sequence and 200 bp of the 3rd exon with 3' ***splice*** ***acceptor*** and polyadenylation site. These sequences are cloned into the plasmid pXF3 contg. the origin of SV40 which allows plasmid amplification in COS cells. To trap exons, genomic fragments (0.5-10 kb) are cloned into a unique Kpn1 site in the intron. Recombinant vectors are transfected by the DEAE method into trypsinized COS cells and after 2 days RNA is analyzed for trapped sequences by cDNA synthesis and PCR using primers from the 5' and 3' insulin exon sequences. To test the method a 475 bp genomic fragment contg. the 330 bp C.delta.3 exon from the IgD was cloned in either the right (pL53InC.delta.3) or wrong (pL53In3.delta.C) orientation into pL53In. Only if the genomic fragment was cloned in the

right orientation was the C.delta.3 exon trapped. A 2.8 kb genomic fragment contg. a single 224 bp exon (ExO) of the uvomorulin gene was cloned in both orientations into pL53In. By transient expression of pL53InExO, which contained the insert in the correct orientation a PCR product of about 450 bp was obtained indicating a trapped exon sequence of the size of ExO (224 bp).

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